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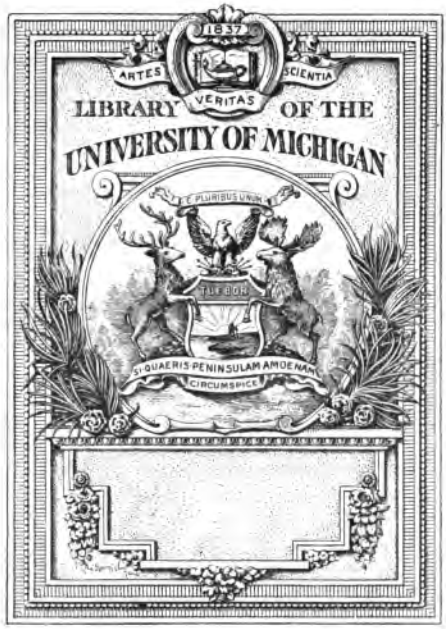
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The Council think it proper to state that the authors of the several communications are alone responsible for the statements, reasonings, and opinions contained in their respective papers.

20, HANOVER SQUARE, W.;
October 11*th*, 1901.

228203

CONTENTS.

PAGE

Notice from the Council v

List of Illustrations . . . . . xvi, xvii

List of Officers and Members of the Council during 1901–2 xix

List of Presidents of the Society from its Formation . xx

List of Honorary Members of the Society . . xxi, xxii

List of Members of the Society xxiii

Report of the Council li

Balance-sheet liii

Presidential Address lv

Papers read:

I. A case of Acute Actinomycosis. By Sir Dyce Duckworth, M.D., and Howard Marsh . 1

II. The Sequel to a case of Hydatid of the Liver: showing also the results of Abdominal Section in Ascites. By Norman Dalton, M.D. 5

PAGE

III. On the Diastolic Cardiac Sound which causes Spurious Reduplication of the Second Sound at the Apex, and is sometimes called the Third Sound of the Heart. By T. Stacey Wilson, M.D. . 10

IV. A case of Thrombosis of the Inferior Vena Cava and of the Iliac and Femoral Veins, of acute onset and fatal termination, with symptoms resembling those of Perforated Gastric Ulcer. By Sidney Phillips, M.D. 24

V. A case of late Syphilitic Pyrexia (Intermittent Essential Fever of Syphilis). By Campbell Williams 28

VI. A case in which Splenectomy was performed for a Wandering Spleen. By J. Bland-Sutton 31

VII. A case of Nephritis without Albuminuria: death in six weeks: no scarlatina: autopsy. By W. P. Herringham, M.D. . 34

VIII. A case of Ankylostomiasis. By W. J. Tyson, M.D. 37

IX. A case of Hæmatoporphyrinuria not due to Sulphonal. By James Calvert, M.D. With a Report on the Urine by A. E. Garrod, M.D. 41

X. A case of Pneumococcal Suppurating Pericarditis treated by incision and drainage: right lobar pneumonia: right empyema: death. By John Fawcett, M.D., and F. J. Steward, M.S. 46

PAGE

XI. A case of Gonorrhœal Rheumatism, with Bursæ filled with Concretions of Urate of Soda. By W. G. Spencer 51

XII. A case of Fatal Pancreatic Cyst of enormous size simulating Ascites. By Sidney Phillips, M.D. 53

XIII. A case of Subacute Combined Degeneration of the Spinal Cord in association with Pernicious Anæmia. By Frederick E. Batten, M.D. 56

XIV. A case of Aortic Regurgitation probably due to Syphilis. By Charles W. Chapman, M.D. 64

XV. A case of Rheumatic Periostitis with Subcutaneous Nodules. By Hugh Thursfield, M.D. 68

XVI. A case of pronounced Spleno-myelogenic Leucocythæmia subsequent to an attack of Malarial Fever. By David Nabarro, M.D. (introduced by George Thin, M.D.) 70

XVII. A paper on Instantaneous Radiography. By Edward W. H. Shenton (introduced by J. H. Bryant, M.D.) 76

XVIII. Some Personal Experiences of the Epidemic of Enteric Fever among the Troops in South Africa, in the Orange River Colony. By H. H. Tooth, M.D., F.R.C.P. 81

XIX. A case of Volvulus of the Ileum with severe Intestinal Hæmorrhage. By J. H. Bryant, M.D. 144

PAGE

XX. A case of Ureteral Calculus simulating Vesical Calculus. By Francis J. Steward, M.S. 149

XXI. A case illustrating an attempt at the Antenatal Treatment of Hæmophilia. By W. H. B. Brook, M.D. 153

XXII. Poisoning from the External Use of Aniline Oil. By StClair Thomson, M.D. . 156

XXIII. A case of Cancer of the Stomach, with unusual vascular and blood changes. By W. Pasteur, M.D., and C. Price Jones, M.B. 160

XXIV. A case of Perforating Gastric Ulcer, causing a subdiaphragmatic abscess which burst through the diaphragm into the lung. By Norman Dalton, M.D. . . 165

XXV. Some cases of Ascites treated by Abdominal Section. By Leonard A. Bidwell . . 171

XXVI. Traumatic Subdural Hæmorrhage occasioning Convulsions on the Sixth Day after the Injury, and successfully treated by Operation. By Raymond Johnson, B.S., and J. Risien Russell, M.D. . . . 179

XXVII. A case of Extreme Dilatation of the Left Auricle of the Heart. By Isambard Owen, M.D., and W. J. Fenton, M.D. . 183

XXVIII. Acute Suppuration of the Thyroid Gland complicating Typhoid Fever. By Rickman J. Godlee, M.S. 189

PAGE

CLINICAL CASES EXHIBITED:

I. A CASE SHOWING THE RESULT OF A PLASTIC OPERATION FOR THE RELIEF OF RECTAL INCONTINENCE LEFT AFTER A MODIFIED KRASKE'S EXCISION OF THE RECTUM FOR CANCER. By J. JACKSON CLARKE, M.B. 195

II. A CASE OF MYOSITIS OSSIFICANS PROGRESSIVA. By J. WALTER CARR, M.D. 199

III. RADICAL CURE OF A CASE OF CHRONIC FRONTAL SINUS EMPYEMA. By HERBERT TILLEY, M.D., F.R.C.S. 201

IV. A CASE OF GROWTH OF THE LEFT TONSILLAR REGION. By STCLAIR THOMSON, M.D. . 203

V. A CASE OF INTRACTABLE ULCERATION OF THE LIPS AND PALATE. By G. R. TURNER . 204

VI. A CASE OF HÆMOPHILIA WITH DISEASE OF THE KNEE-JOINT (WITH RADIOGRAPHS). By PERCY KIDD, M.D. 205

VII. A CASE OF MULTIPLE CUTANEOUS CARCINOMATOSIS AFTER CARCINOMA MAMMÆ. By H. D. ROLLESTON, M.D. 206

VIII. A CASE OF CYSTIC KIDNEYS, APPARENTLY CONGENITAL. By NORMAN DALTON, M.D. . . 208

IX. A CASE OF TRANSFACIAL BULLET WOUND. By H. BATTY SHAW, M.D. 210

X. A CASE OF HÆMORRHAGE INTO THE SPINAL CORD. By W. A. MARRIS, M.D. (FOR C. E. BEEVOR, M.D.) 212

PAGE

XI. A case of Central Sarcoma of the Lower Jaw three and a half years after Enucleation. By Charters J. Symonds, M.S. 213

XII. Oöphorectomy for Recurrent Mammary Cancer. By Stanley Boyd, B.S. . . 214

XIII. A case of Intractable Ulcer of the Forearm of undetermined origin. By W. Bruce Clarke 214

XIV. Generalised Scleroderma with Raynaud's Disease. By H. D. Rolleston, M.D., and S. Vere Pearson, M.D. 215

XV. A case of Myasthenia Gravis. By Risien Russell, M.D. 216

XVI. A case of Elbow three years excised in a peculiar and unexpected manner for stiffness following fracture: unusually strong result. By Rushton Parker . 218

XVII. A case of Localised Muscular Rigidity. By F. E. Batten, M.D. 219

XVIII. A case of Aortic Regurgitation apparently due to rupture of a healthy valve. By Lauriston E Shaw, M.D. 220

XIX. A case of Dextrocardia, probably congenital. By A. M. Gossage, M.D. . . . 220

XX. A case of unusually great Dilatation of the Heart. By J. Porter Parkinson . 221

XXI. A case of Combined Sclerosis of the Spinal Cord. By G. Newton Pitt, M.D. . . 223

PAGE

XXII. A SIMPLE FORM OF ELECTRIC LIGHT AND HEAT BATH. By F. C. EVE, M.D. 225

XXIII. A CASE OF MULTIPLE FIBRO-NEUROMATA. By C. E. BEEVOR, M.D. 226

XXIV. A CASE OF SYMMETRICAL TUMOURS OF THE LEGS. By J. STEWART RICHARDS, M.D. . 227

XXV. A CASE OF CHRONIC LYMPHANGITIS. By A. E. BRIDGER, M.D. (INTRODUCED BY C. S. DE SEGUNDO, M.D.) 229

XXVI. A CASE OF CYSTIC HYGROMA OF THE NECK, PROBABLY CONGENITAL. By STCLAIR THOMSON, M.D. 231

XXVII. A CASE OF EXTRAORDINARY CURVATURE OF THE LOWER END OF THE FEMUR, FOLLOWING EPIPHYSITIS. By J. S. RICHARDS, M.D., B.S. . 231

XXVIII. A CASE OF PROGRESSIVE MYOSITIS OSSIFICANS. By H. D. ROLLESTON, M.D. (FOR F. W. BURTON FANNING, M.D.) 232

XXIX. A CASE OF PECULIAR AFFECTION OF THE JOINTS. By HUGH THURSFIELD, M.D. (FOR A. E. GARROD, M.D) 233

XXX. A CASE OF ENLARGEMENT OF THE RIGHT FOOT: ? CAUSE. By H. BETHAM ROBINSON . . 234

XXXI. A CASE OF ACTINOMYCOSIS IN PAROTID REGION. By RAYMOND JOHNSON 235

XXXII. A CASE OF ACUTE BRONCHOCELE. By SIDNEY PHILLIPS, M.D. 236

XXXIII. A CASE OF TACHYCARDIA (? GRAVES'S DISEASE). By SIDNEY PHILLIPS, M.D. 237

PAGE

XXXIV. A case of Injury to Left Optic Nerve. By R. J. Godlee, M.S. 237

XXXV. A case of Congenital Morbus Cordis: ? Patent Ductus Arteriosus Regurgitation. By Percy Kidd, M.D. . . . 238

XXXVI. A case of Bullet Wound of the Brain, showing injuries to the Optic Nerve and the Pyramidal Tract, corresponding to the course and present position of the Bullet. By W. G. Spencer . . . 238

XXXVII. Normal Thoracic Resonance in Left-handed Persons. By Sir Hugh Beevor, Bart., M.D. 242

XXXVIII. Unusual case of Obesity in a Child. By G. F. Still, M.D. (introduced by Dr. Dalton) 243

XXXIX. Sequel of a case of Myxœdema which was under observation at intervals for twenty-three years. By Sir Dyce Duckworth, M.D. 244

XL. Retro-collic Spasm. By C. E. Beevor, M.D. 245

XLI. A case of Lymphadenoma with Growths in the Skin. By Lee Dickinson, M.D. . . 246

XLII. Congenital Morbus Cordis: ? Patent Ductus Arteriosus. By Percy Kidd, M.D. . . 248

XLIII. Myxœdema in a girl æt. 15. By Percy Kidd, M.D. 249

XLIV. A case of Chronic Hypertrophic Osteo-arthropathy. By A. B. Soltau, M.B. (introduced by Percy Kidd, M.D.) . . 249

PAGE

XLV. Inveterate Ulceration of both Hands. By J. Hutchinson, jun. 251

XLVI. Dislocation of the Patella. By Charters Symonds, M.S. 252

XLVII. Hæmorrhagic Gummatous Myositis. By Charters Symonds, M.S. 252

XLVIII. Knee-joint Eight Years after Erasion. By Charters Symonds, M.S. 253

XLIX. Sequel to a case of Myasthenia gravis pseudo-paralytica (Strümpell's Asthenic Bulbar Paralysis). By Leonard Guthrie, M.D. 253

INDEX 257

LIST OF ILLUSTRATIONS.

PLATES.

TO PAGE PAGE

PLATES I—IV. ILLUSTRATING CASE OF SUBACUTE COMBINED DEGENERATION OF THE SPINAL CORD. FREDERICK E. BATTEN, M.D. 62

PLATES V—VII. ILLUSTRATING PAPER ON INSTANTANEOUS RADIOGRAPHY. EDWARD W. H. SHENTON . . . 80

PLATES VIII, IX. ILLUSTRATING PAPER ON CANCER OF THE STOMACH. W. PASTEUR, M.D., AND C. PRICE-JONES, M.B. 164

PLATE X. ILLUSTRATING CASE OF HÆMOPHILIA WITH DISEASE OF THE KNEE-JOINT. PERCY KIDD, M.D. . . 206

PLATE XI. ILLUSTRATING CASE OF CURVATURE OF THE LOWER END OF THE FEMUR. J. S. RICHARDS, M.D. . 232

PLATE XII. ILLUSTRATING CASE OF ENLARGEMENT OF THE RIGHT FOOT. H. BETHAM ROBINSON 234

PLATE XIII. ILLUSTRATING CASE OF BULLET WOUND OF THE BRAIN. W. G. SPENCER 242

PLATE XIV. ILLUSTRATING CASE OF LYMPHADENOMA. LEE DICKINSON, M.D. 248

PLATE XV. ILLUSTRATING CASE OF MYXŒDEMA. PERCY KIDD, M.D. 249

PLATES XVI—XVIII. ILLUSTRATING CASE OF OSTEO-ARTHROPATHY. A. B. SOLTAU, M.B. 250

ILLUSTRATIONS IN TEXT.

PAGE

FIGS. 1—14. CHARTS ILLUSTRATING SPURIOUS REDUPLICATION OF THE SECOND SOUND AT THE APEX. T. STACEY WILSON, M.D. 13, 17, 19, 20, 21, 22, 23

CHART ILLUSTRATING CASE OF LATE SYPHILITIC PYREXIA. CAMPBELL WILLIAMS 29

CHART ILLUSTRATING CASE OF PNEUMOCOCCAL SUPPURATING PERICARDITIS. JOHN FAWCETT, M.D., AND F. J. STEWARD 49

FIG. 15. ILLUSTRATING PAPER ON INSTANTANEOUS RADIOGRAPHY. EDWARD W. H. SHENTON 77

FIG. 16. ILLUSTRATING CASE OF VOLVULUS OF THE ILEUM WITH SEVERE INTESTINAL HÆMORRHAGE. J. H. BRYANT, M.D. 146

FIGS. 17, 18. ILLUSTRATING CASE OF PERFORATING GASTRIC ULCER. NORMAN DALTON, M.D. 167, 169

FIG. 19. ILLUSTRATING CASE OF ACUTE SUPPURATION OF THE THYROID GLAND, COMPLICATING TYPHOID FEVER. R. J. GODLEE 190

CHART ILLUSTRATING ABOVE CASE. R. J. GODLEE . . 191

FIGS. 20—24. ILLUSTRATING CASE SHOWING RESULT OF A PLASTIC OPERATION FOR THE RELIEF OF RECTAL INCONTINENCE. J. JACKSON CLARKE 197

FIG. 25. ILLUSTRATING CASE OF MYOSITIS OSSIFICANS PROGRESSIVA. J. WALTER CARR, M.D. 199

FIGS. 26, 27. ILLUSTRATING CASE OF MULTIPLE CUTANEOUS CARCINOMATOSIS. H. D. ROLLESTON, M.D. . . 207

FIG. 28. ILLUSTRATING CASE OF UNUSUALLY GREAT DILATATION OF THE HEART. J. PORTER PARKINSON, M.D. 222

PERIMETER CHART ILLUSTRATING CASE OF BULLET WOUND OF THE BRAIN. W. G. SPENCER 239

FIG. 29. ILLUSTRATING ABOVE CASE. W. G. SPENCER . 240

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1891 BARTON, J. KINGSTON, 2, Courtfield Road, S.W. (C. 1901– .)

O.M. BARWELL, RICHARD, 55, Wimpole Street, W. (C. 1872–5, V.P. 1877–9.) *Trans.* 21, *C.C.* 2.

O.M. **BASTIAN**, HENRY CHARLTON, M.D., F.R.S., 8A, Manchester Square, W. (C. 1876–8, V.P. 1891–3.) *Trans.* 7.

1896 BATTEN, FREDERICK E., M.D., 124, Harley Street, W. *Trans.* 3, *C.C.* 2.

1899 BATTEN, RAYNER DERRY, M.D., B.S., Campden Lodge, Campden Hill Road, W.

1888 BATTERHAM, JOHN WILLIAMS, M.B., B.S., Bank House, Grand Parade, St. Leonard's.

1886 BATTLE, WILLIAM HENRY, 49, Harley Street, W. (C. 1899– .) *Trans.* 7, *C.C.* 12.

1868 BÄUMLER, CHRISTIAN G. H., M.D., University of Erlangen. *Trans.* 4.

1896 BEATON, ROBERT MILNE, M.B., 7, Dartmouth Park Road, N.W.

1898 BEDDARD, ARTHUR PHILIP, M.B., 44, Seymour Street, Portman Square, W.

1880 **BEEVOR**, CHARLES EDWARD, M.D., 135, Harley Street, W. (C. 1890–1.) *Trans.* 2, *C.C.* 9.

1901 BEEVOR, SIR HUGH REEVE, Bart., M.D., 17, Wimpole Street, Cavendish Square, W. *C.C.* 1.

1884 BENHAM, F. LUCAS, M.D., Campden House, nr. Exeter, Semaphore, South Australia. *Trans.* 4.

1883 BENHAM, ROBERT FITZROY, 11, Earl's Court Square, S.W.

1874 BENNETT, WILLIAM HENRY, 1, Chesterfield Street, W. (C. 1889, 1892–4, S. 1890–1, V.P. 1895–8.) *Trans.* 4, *C.C.* 3.

1896 BENTHAM, EDWARD COOPER, 10, Princes Street, Cavendish Square, W.

1889 BENTLEY, ARTHUR J. M., M.D., Mena House, Pyramids, Cairo, Egypt.

1896 BERKELEY, G. H. A. COMYNS, M.B., 53, Wimpole Street, W.

1882 BERRY, FREDERICK HAYCRAFT, M.B., Watford, Herts.

ELECTED

1885 BERRY, JAMES, B.S., 21, Wimpole Street, W. (C. 1898–1901.)

1889 BIDWELL, LEONARD ARTHUR, 59, Wimpole Street, W. (C. 1901– .) *Trans.* 5, *C.C.* 1.

1882 BINDLEY, PHILIP HENRY, M.B. (Travelling).

1889 BISSHOPP, FRANCIS ROBERT BRYANT, M.A., M.B., B.S., Belle Vue, Mount Pleasant, Tunbridge Wells.

1887 BLAND-SUTTON, JOHN, 48, Queen Anne Street, W. (C. 1894-7.) *Trans.* 13. *C.C.* 3.

1893 BOND, JAMES WILLIAM, M.D., 26, Harley Street, W. *C.C.* 2.

1898 BOSANQUET, WILLIAM CECIL, M.D., 117A, Harley Street, Cavendish Square, W.

1888 BOSTOCK, ROBERT ASHTON, Cefn Mor, Penmaen, Glamorganshire.

1900 BOUSFIELD, ARTHUR, M.B., The Cheal, Hornsey Lane, N.

1899 BOWLAN, MARCUS MARWOOD, M.B., B.S., The Infirmary, Raine Street, St. George-in-the-East, E.

1883 BOWLBY, ANTHONY A., C.M.G. (*Hon. Secretary*), 24, Manchester Square, W. (C. 1891–3.) *Trans.* 3. *C.C.* 3.

1883 Bowles, ROBERT LEAMON, M.D., 16, Upper Brook Street, W. (C. 1890–1.)

1896 BOX, CHARLES R., M.D., 2, Devonshire Place, Portland Place, W. *Trans.* 1.

1889 BOYD, STANLEY, M.B., 134, Harley Street, W. (C. 1899–.) *Trans.* 1, *C.C.* 7.

1890 BRADFORD, JOHN ROSE, M.D., F.R.S., 8, Manchester Square, W. (C. 1900– .) *Trans.* 2.

1878 BRIDGES, ROBERT, M.B., M.A., The Manor House, Yattendon, Berkshire. *Trans.* 1.

1868 BRIGHT, GEORGE CHARLES, M.D., Cannes, Alpes Maritimes, France.

O.M. BROADBENT, Sir WILLIAM HENRY, Bart., M.D., F.R.S., 84, Brook Street, W. (C. 1871–3, V.P. 1881–3, P. 1887–8.) *Trans.* 17.

1887 BROCK, J. H. E., M.D., B.S., 77, Fellows Road, South Hampstead, N.W.

1894 BRODIE, C. GORDON, Fernhill, Wootton Bridge, Isle of Wight. *C.C.* 1.

1898 Brook, WILLIAM HENRY BREFFIT, M.D., B.S., 8, Eastgate, The Close, Lincoln. *Trans.* 1.

1890 BROWN, WALTER HENRY, 19, Queen Street, Leeds. *Trans.* 2.

1876 BROWNE, GEORGE BUCKSTON, 80, Wimpole Street, W. (C. 1891–3.) *Trans.* 3.

1893 BROWNE, JAMES WILLIAM, M.B., 37, Holland Park Avenue, W. *Trans.* 1.

1887 BROWNE, OSWALD AUCHINLECK, M.A., M.D., 7, Upper Wimpole Street, W.

1883 BRUCE, JOHN MITCHELL, M.D., 23, Harley Street, W. (C. 1901– .)

1893 BRYANT, JOHN HENRY, M.D., 4, St. Thomas's Street, S.E., and 8, Mansfield Street, Portland Place, W. *Trans.* 2.

ELECTED

O.M. BRYANT, THOMAS, M.Ch., 27, Grosvenor Street, W. (C. 1872, V.P. 1876–7, P. 1885–6.) *Trans.* 8.

1890 BUCKLAND, FRANCIS O., B.A., M.B., C.M., Brockenhurst, Hants. *Trans.* 1.

1901 BUCKNALL, THOMAS RUPERT HAMPDEN, M.S., University College Hospital, W.C.

1891 BURGHARD, FREDERIC FRANÇOIS, M.D., M.S., 86, Harley Street, W.

1881 BURNET, ROBERT WILLIAM, M.D., 36, Grosvenor Street, W. *Trans.* 1.

1879 BURTON, WILLIAM EDWARD, 24, Wimpole Street, W.

1900 BUSCH, JOSEF PAUL ZUM, M.D., 12, Finsbury Square, E.C.

1881 BUTLIN, HENRY TRENTHAM, 82, Harley Street, W. (C. 1887–9.) *Trans.* 7, *C.C.* 1.

1884 BUXTON, DUDLEY WILMOT, M.D., B.S., 82, Mortimer Street, W.

O.M. BUZZARD, THOMAS, M.D., 74, Grosvenor Street, W. (S. 1870–2, C. 1873–6, V.P. 1880–1, P. 1895–7.) *Trans.* 15, *C.C.* 3.

1901 CADDY, ADRIAN, M.B., Parish Infirmary, Brownlow Hill, Liverpool.

1891 CADDY, ARNOLD, 2/2, Harington Street, Calcutta.

1886 CAHILL, JOHN, M.D., 12, Seville Street, Lowndes Square, S.W. *C.C.* 1.

1893 CALEY, HENRY ALBERT, M.D., 24, Upper Berkeley Street, W. *C.C.* 1.

1890 CALVERT, JAMES, M.D., The Warden's House, St. Bartholomew's Hospital, E.C. *Trans.* 2, *C.C.* 2.

1893 CAMPBELL, HARRY, M.D., 23, Wimpole Street, W., and Coniston Cottage, Fox Lane, Upper Norwood, S.E. *C.C.* 3.

1891 CARLESS, ALBERT, M.S., M.B., 10, Welbeck Street, W.

1890 CARR, JOHN WALTER, M.D., 19, Cavendish Place, W. *Trans.* 1, *C.C.* 5.

1901 CARSON, HERBERT WILLIAM, 55A, Welbeck Street, Cavendish Square, W.

1885 CATON, RICHARD, M.D., 86, Rodney Street, Liverpool. *Trans.* 3.

O.M. CAYLEY, WILLIAM, M.D., 27, Wimpole Street, W. (C. 1874–5, S. 1876–8, C. 1879–80, V.P. 1885–6.) *Trans.* 8, *C.C.* 3.

1890 CHAPMAN, CHARLES WILLIAM, M.D., 21, Weymouth Street, W. (C. 1900– .) *Trans.* 2, *C.C.* 2.

1884 CHAPMAN, PAUL M., M.D., 1, St. John's Street, Hereford. *C.C.* 1.

1893 CHARSLEY, ROBERT STEPHEN, "The Old Barn," Slough.

1885 CHEYNE, W. WATSON, M.B., C.M., F.R.S., 75, Harley Street, W. (C. 1894–6.) *Trans.* 2, *C.C.* 8.

1873 CHISHOLM, EDWIN, M.D., Abergeldie, Ashfield, near Sydney, New South Wales.

O.M. CHURCH, Sir WILLIAM SELBY, Bart., M.D., 130, Harley Street, W. (C. 1874–6.)

1873 CHURTON, THOMAS, M.D., 35, Park Square, Leeds. (C. 1889–91.) *Trans.* 6.

O.M. CLAPTON, EDWARD, M.D., Towercroft, 41, Eltham Road, Lee, S.E. (C. 1872–4.) *Trans.* 1.

1874 CLARK, ANDREW, 71, Harley Street, W. *Trans.* 1.

1887 CLARK, FRANCIS WILLIAM, Hong-Kong.

ELECTED

1898 CLARKE, JAMES JACKSON, M.B., 18, Portland Place, W. *C.C.* 5.

1885 Clarke, J. MICHELL, M.B., 28, Pembroke Road, Clifton, Bristol.

1885 CLARKE, WILLIAM BRUCE, M.B., 51, Harley Street, W. (C. 1894–7.) *Trans.* 2, *C.C.* 2.

1877 Clay, ROBERT HOGARTH, M.D., 4, Windsor Villas, Plymouth.

1887 CLEMOW, ARTHUR HENRY WEISS, M.D., C.M., 101, Earl's Court Road, W. *Trans.* 1, *C.C.* 1.

1877 CLUTTON, HENRY HUGH, M.A., 2, Portland Place, W. (C. 1885–7.) *Trans.* 12, *C.C.* 3.

1898 COBB, ERNEST HERBERT, Belmont, Stevenage, Herts.

1893 COLE, ROBERT HENRY, M.D., Moorcroft, Hillingdon, Middlesex.

1882 COLLIER, HERBERT, M.D., The Grange, Gorleston, Great Yarmouth, Norfolk.

1896 COLMAN, WALTER STACY, M.D., 22, Wimpole Street, W. *Trans.* 2, *C.C.* 3.

1882 COLQUHOUN, DANIEL, M.D., Dunedin, New Zealand.

1868 COOPER, FRANK W. (address uncommunicated).

1899 COOPER, HARRY, M.D., Fownhope, Surbiton Hill, Surrey.

1880 COTTLE, WYNDHAM, M.D., 39, Hertford Street, W.

O.M. COUPER, JOHN, 80, Grosvenor Street, W. (C. 1874.)

1875 COUPLAND, SIDNEY, M.D., 16, Queen Anne Street, W. (S. 1882–4, C. 1885–7, V.-P. 1898–1901.) *Trans.* 5, *C.C.* 3.

1886 COUSINS, JOHN WARD, M D., Riversdale, Kent Road, Southsea. *Trans.* 1.

1882 COXWELL, C. F., M.B., Brighton, Melbourne, Victoria. *Trans.* 2.

1897 CRAWFURD, RAYMOND H. P., M.D., 71, Harley Street, Cavendish Square, W. *C.C.* 2.

1879 CRIPPS, WILLIAM HARRISON, 2, Stratford Place, W. (C. 1886–8.) *Trans.* 3.

1872 CRITCHETT, G. ANDERSON, 21, Harley Street, W.

1877 CROCKER, HENRY RADCLIFFE, M.D., 121, Harley Street, W. (C. 1884–5.) *Trans.* 16.

1896 CROOKE-LAWLESS, W. R., Surg.-Maj. A.M.S., M.D., Guards' Club, S.W.

1896 CROSSE, WILLIAM HENRY, M.D., 45, Dover Street, W.

1898 CROUCH, HERBERT CHALLICE, 31, Welbeck Street, Cavendish Square, W.

1890 CROWLE, THOMAS HENRY RICKARD, 35, St. James's Place, S.W.

1893 CURTIS, HENRY JONES, M.D., B.S., 30, Harley Street, W. *C.C.* 1.

1872 DALBY, Sir WILLIAM BARTLETT, M.B., 18, Savile Row, W. (C. 1879–81.) *Trans.* 4.

1896 DALTON, NORMAN, M.D., 4, Mansfield Street, Portland Place, W. *Trans.* 2, *C.C.* 6.

1898 DARLEY-HARTLEY, WILLIAM, M.D., Park House, Kloof Street, Cape Town, South Africa.

1893 DAUBER, JOHN HENRY, M.B., B.Ch., 29, Charles Street, Berkeley Square, W.

Elected

1886 Davies, Arthur T., M.D., 23, Finsbury Square, E.C. (C.1897–1900.) *C.C.* 13.

1879 Davy, Henry, M.D., 29, Southernhay, Exeter.

1889 Dean, Henry Percy, M.B., 69, Harley Street, W.

1897 Deane, John Henry, 50, Wallwood Road, Leytonstone, Essex.

1899 De Chazal, Edmond Lucien, M.D., Port Louis, Mauritius.

1879 Dennis, Frederic S., M.D., 542, Madison Avenue, New York, U.S.A.

1875 Dent, Clinton T., 61, Brook Street, W. (C. 1884–6.) *Trans.* 5. *C.C.* 1.

O.M. Dickinson, William Howship, M.D., 9, Chesterfield Street, W. (C. 1874–5, V.P. 1888–9.) *Trans.* 2.

1891 Dickinson, William Lee, M.D., 9, Chesterfield Street, W. *Trans.* 7, *C.C.* 1.

1894 Dickson, Thomas Hugh, M.B., B.C., H.M. Customs, Lower Thames Street, E.C.

1871 Diver, Ebenezer, M.D., "Garstone," St. Margaret's-on-Thames, Twickenham. (C. 1890–2.) *Trans.* 1.

1868 Drage, Charles, M.D., Hatfield, Herts.

1896 Drew, Douglas, M.D., B.S., 39, Devonshire Street, Portland Place, W.

1879 Drewitt, F. G. Dawtrey, M.D., 14, Palace Gardens Terrace, Kensington, W. (C. 1886–8.) *Trans.* 2.

O.M. Duckworth, Sir Dyce, M.D., LL.D. 11, Grafton Street, Bond Street, W. (C. 1875–7, V.P. 1887–8, P. 1891–2.) *Trans.* 20, *C.C.* 7.

1884 Duke, Edgar, 11, Wilbury Road, Hove.

1869 Duke, Olliver Thomas, M.B., Surgeon, Bengal Army, India.

1899 Duncan, Andrew, M.D., 24, Chester Street, Grosvenor Place, S.W.

1889 Duncan, John, M.D., St. Petersburg.

1889 Dunn, Louis Albert, M.S., The College, Guy's Hospital, S.E. (C. 1899– .) *Trans.* 2, *C.C.* 2.

1884 Edmunds, Walter, M.C., 2, Devonshire Place, Portland Place, W. *Trans.* 2.

1899 Eïd, Alfred, M.D., Cairo, Egypt.

1882 Emond, Emile, M.D., 113, Boulevard Beaumarchais, Paris.

1868 Evans, Julian, M.B., 123, Finborough Road, Redcliffe Square, S.W.

1893 Evans, Willmott H., 2, Upper Wimpole Street, Cavendish Square, W. *C.C.* 1.

1888 Eve, Frederic S., 125, Harley Street, Cavendish Square, W. (C. 1899– .) *Trans.* 4, *C.C.* 10.

1877 Ewart, William, M.D., 33, Curzon Street, W. (C. 1884–6.) *Trans.* 2, *C.C.* 2.

1899 Fagge, Charles Herbert, M.S., 22, St. Thomas's Street, London Bridge, S.E.

1868 Fairbank, Frederick Royston, M.D., Westcott, Dorking, Surrey. *Trans.* 2.

1889 Fardon, Edward Ashby, Middlesex Hospital, W.

1900 Fawcett, John, M.D., 24, St. Thomas's Street, London Bridge, S.E. *Trans.* 1.

ELECTED

1885 FENN, EDWARD LIVEING, M.D., Grey Friars, Colchester.
1887 FENWICK, E. HURRY, 14, Savile Row, W. *Trans.* 2, *C.C.* 2.
1872 FENWICK, J. C. J., M.D., Long Framlington, Morpeth.
1893 FENWICK, WILLIAM SOLTAU, M.D., 8, Devonshire Street, W. *Trans.* 2.
1878 FIELD, GEORGE P., 34, Wimpole Street, W.
1876 FINLAY, DAVID WHITE, M.D., 2, Queen's Terrace, Aberdeen. (C. 1885–7, S. 1891.) *Trans.* 7.
1894 FLETCHER, HERBERT MORLEY, M.D., 98, Harley Street, W. *Trans.* 1.
1878 **FONMARTIN**, HENRY DE, M.D., 26, Newberry Terrace, Lower Bullar Street, Nichols Town, Southampton.
1889 FORBES, DANIEL MACKAY, Shoreditch Infirmary, 204, Hoxton Street, N.
1890 FOSTER, MICHAEL G., M.B., M.A., Villa Camilla, San Remo, Italy.
1898 FOWLER, CHARLES OWEN, M.D., Cotford House, Thornton Heath, Surrey.
1881 FOWLER, JAMES KINGSTON, M.D., 35, Clarges Street, W. (C. 1887–9, 1899–, S. 1896–9.) *Trans.* 4, *C.C.* 6.
1886 FOX, R. HINGSTON, M.D., 23, Finsbury Square, E.C. *C.C.* 1.
1878 FOX, THOMAS COLCOTT, M.B., 14, Harley Street, W. (C. 1888–90.) *Trans.* 7, *C.C.* 2.
1893 FOXWELL, ARTHUR, M.D., 22, Newhall Street, Birmingham.
1898 FRANK, PHILIP, M.D., 3, Elvaston Place, Queen's Gate, S.W.
1897 FRIPP, ALFRED DOWNING, C.B., M.S., 19, Portland Place, W. *Trans.* 3.
1899 FÜRTH, KARL, M.D., 94, Harley Street, W.
1890 FULLER, HENRY ROXBURGH, M.D., 45, Curzon Street, W.
1897 FURNIVALL, PERCY, 28, Weymouth Street, Portland Place, W.
1891 FYFFE, W. KINGTON, M.B., B.C., 19, Duke Street, Manchester Square, W. (Travelling, New Zealand.) *Trans.* 1.
1888 GAGE-BROWN, CHARLES HERBERT, M.D., 85, Cadogan Place, S.W.
1895 GALLOWAY, JAMES, M.D., 54, Harley Street, W.
1887 GARROD, ARCHIBALD EDWARD, M.A., M.D., 9, Chandos Street, Cavendish Square, W. (C. 1898–1901.) *Trans.* 4, *C.C.* 3.
1879 GARSTANG, THOMAS WALTER HARROPP, Englefield, Delamer Road, Bowdon, Cheshire.
1885 GIBBONS, ROBERT ALEXANDER, M.D., 29, Cadogan Place, S.W. *Trans.* 1.
1893 GIBBS, CHARLES, 115, Harley Street, W.
1875 GILBART-SMITH, T., M.A., M.D., 68, Harley Street, W. (C. 1883–5.)
1868 GLOVER, JAMES GREY, M.D., 25, Highbury Place, N. (C. 1878–80, V.P. 1892–4.) *Trans.* 2.
1893 GLOVER, LEWIS G., M.B., B.C., 17, Belsize Park, Hampstead, N.W.
1875 GODLEE, RICKMAN JOHN, M.S., 19, Wimpole Street, W. (C. 1882–3, 1887–9, S. 1884–6, V.P. 1895–8.) *Trans.* 14, *C.C.* 9.
1901 GOFFE, ERNEST GEORGE LEOPOLD, Brook Hospital, Shooter's Hill, Kent.
1882 GOLDIE, ROBERT WILLIAM, Hirst House, Hirst, Morpeth, Northumberland.

ELECTED

1878 GOLDING-BIRD, C. H., M.B., 12, Queen Anne Street, W. (C. 1887–9.) *Trans.* 17, *C.C.* 1.

1894 GOODALL, EDWARD WILBERFORCE, M.D., Eastern Hospital, Homerton, N.E. *Trans.* 5.

1875 GOODHART, JAMES FREDERIC, M.D., 25, Portland Place, W. (C. 1880–2, V.P. 1897–1900.) *Trans.* 14, *C.C.* 2.

1891 GOODMAN, ROGER NEVILLE, M.B., 3, Grove Crescent, Kingston-on-Thames.

1869 GOODRIDGE, HENRY FREDERICK AUGUSTUS, M.D., 10, Brock Street, Bath.

1882 GOODSALL, D. H., 17, Devonshire Place, W.

1877 GOULD, A. PEARCE, M.S., 10, Queen Anne Street, W. (C. 1885–7, 1895–8, S. 1892–5, V.-P. 1898–1901.) *Trans.* 14, *C.C.* 9.

1875 GOWERS, Sir WILLIAM RICHARD, M.D., F.R.S., 50, Queen Anne Street, W. (C. 1881–2.) *Trans.* 5.

1891 GRANT, J. DUNDAS, M.D., 18, Cavendish Square, W.

1868 GREEN, T. HENRY, M.D., 74, Wimpole Street, W. (C. 1877–9.) *Trans.* 2, *C.C.* 1.

1875 Greenfield, WILLIAM SMITH, M.D., 7, Heriot Row, Edinburgh. (C. 1881.) *Trans.* 3.

1893 GRIFFITH, WALTER SPENCER ANDERSON, M.D., 96, Harley Street, W.

1895 Grube, KARL, M.D., Neuenahr, Germany. *Trans.* 1.

1895 GUTHRIE, LEONARD GEORGE, M.D., 15, Upper Berkeley Street, W. *C.C.* 6.

1887 HABERSHON, SAMUEL HERBERT, M.D., 88, Harley Street, W. (C. 1901– .) *C.C.* 1.

1900 HADLEY, WILFRED J., M.D., 58, Harley Street, Cavendish Square, W.

1875 HALE, C. D. B., 3, Sussex Place, W. *Trans.* 1.

1878 HALL, F. DE HAVILLAND, M.D., 47, Wimpole Street, W. (C. 1885–7.) *Trans.* 8, *C.C.* 5.

1889 HALSTEAD, GEORGE EZRA, M.D., B.S., Albion Hill House, Ramsgate.

1888 HANDFIELD-JONES, MONTAGU, M.D., 35, Cavendish Square, W.

1886 Handford, HENRY, M.D., 6, Regent Street, Nottingham. (C. 1893–4.) *Trans.* 8, *C.C.* 1.

1886 HARDIE, JAMES, M.D., 15, St. John Street, Manchester.

1890 HARPER, JAMES, M.D., 25, Rosary Gardens, South Kensington, S.W.

1872 HARRIS, HENRY, M.D., Trengweath, Redruth, Cornwall.

1889 HARRIS, HERBERT ELWIN, M.B., 13, Lansdown Place, Victoria Square, Clifton, Bristol. *Trans.* 1.

1881 HARRISON, CHARLES EDWARD, M.B., Grenadier Guards Hospital, Rochester Row, S.W.

1892 HARRISON, DAMER, 53, Rodney Street, Liverpool. *Trans.* 1.

1869 HAWARD, J. WARRINGTON, 57, Green Street, Grosvenor Square, W. (C. 1876–8, 1884–6, S. 1881–3, V.P. 1888.) *Trans.* 14.

1899 Hawkes, CLAUDE SOMERVILLE, Glencairn, Wickham Terrace, Brisbane, Queensland, Australia.

ELECTED

1886 HAWKINS, FRANCIS HENRY, M.D., 73, London Street, Reading. *Trans.* 4, *C.C.* 3.

1889 HAWKINS, HERBERT PENNELL, M.D., B.Ch.; 56, Portland Place, W. (C. 1900- .) *Trans.* 1.

1890 HAWKINS-AMBLER, GEORGE ARTHUR, 30, Rodney Street, Liverpool.

1900 HAYWARD, JOHN ARTHUR, M.D., 17, Lingfield Road, Wimbledon, S.W.

1879 HENDERSON, GEORGE COURTENAY, M.D., Kingston, Jamaica, West Indies.

1882 HERON, GEORGE ALLAN, M.D., 57, Harley Street, W.

1884 HERRINGHAM, WILMOT PARKER, M.D., 40, Wimpole Street, W. (C. 1894–7.) *Trans.* 6, *C.C.* 3.

1888 Hetherington, GEORGE HAYNES, 10, Museum Street, Ipswich.

1897 HILLIER, ALFRED P., M.D., C.M., 30, Wimpole Street, W.

1874 HOLDERNESS, WILLIAM BROWN, 15, Park Street, Windsor.

1868 Holman, CONSTANTINE, M.D., 26, Gloucester Place, Portman Square, W. (C. 1894–7.)

O.M. HOLMES, TIMOTHY, 6, Sussex Place, Hyde Park, W. (C. 1867–9, V.P. 1873–5.) *Trans.* 16.

1878 HOOD, DONALD WILLIAM CHARLES, M.D., 43, Green Street, Park Lane, W. (C. 1891–3.) *Trans.* 1.

1884 HORSLEY, VICTOR, M.B., F.R.S., 25, Cavendish Square, W. (C. 1894.) *Trans.* 2, *C.C.* 1.

1901 HORTON-SMITH, PERCIVAL, M.D., 15, Upper Brook Street, Grosvenor Square, W.

1895 HOUGH, CHARLES HENRY, Full Street, Derby.

1880 HOVELL, T. MARK, 105, Harley Street, W. *Trans.* 1.

1893 HOWARD, R. J. BLISS, M.D., 31, Queen Anne Street, W.

1876 HOWSE, HENRY GREENWAY, M.S., 59, Brook Street, W. (C. 1881–3, V.P. 1890–2.) *Trans.* 3.

1898 HULKE, SYDNEY BACKHOUSE, 162, Holland Road, Kensington, W.

1897 HUMPHRYS, CHARLES B., Eagle House, Blandford, Dorset.

1897 HUNT, GEORGE BERTRAM, M.B., 47, Albemarle Crescent, Scarborough. *Trans.* 1, *C.C.* 1.

1892 HUNTER, WILLIAM, M.D., 103, Harley Street, W.

O.M. HUTCHINSON, JONATHAN, F.R.S., 15, Cavendish Square, W. (C. 1867–8, V.P. 1875–6.) *Trans.* 14, *C.C.* 1.

1896 HUTCHINSON, JONATHAN, jun., 15, Cavendish Square, W. *Trans.* 3, *C.C.* 4.

1893 IONIDES, THEODORE HENRY, M.B., B.S., 30, Palmeira Square, West Brighton.

1883 JACKSON, GEORGE HENRY, Ashburton, Carew Road, Eastbourne.

O.M. JACKSON, J. HUGHLINGS, M.D., F.R.S., 3, Manchester Square, W. (C. 1872–3, V.P. 1887.) *Trans.* 2, *C.C.* 1.

ELECTED

1877 JACOBSON, WALTER HAMILTON ACLAND, M.B., M.Ch., 66, Great Cumberland Place, W. (C. 1890–2.) *Trans.* 2, *C.C.* 1.

1888 JAMES, JAMES THOMAS, M.D., 30, Harley Street, W.

1901 JOHNSON, EDWARD ANGAS, M.B., B.S., St. Catherine's, Prospect, South Australia.

1889 JOHNSON, RAYMOND, M.B., B.S., 11, Wimpole Street, W. (C. 1900– .) *Trans.* 5, *C.C.* 3.

1893 JOHNSTON, G. F., M.D., 3, Montagu Place, Montagu Square, W.

1878 JOHNSTON, WILLIAM, M.D., M.C., 16, Lonsdale Terrace, Upper Kent Street, Leicester.

1898 Jones, ROBERT, 11, Nelson Street, Liverpool. *Trans.* 2.

1872 JONES, THOMAS RIDGE, M.D., 4, Chesham Place, S.W. (C. 1892–3.)

1876 JORDAN, FURNEAUX, Harborne, Birmingham. *Trans.* 1.

1886 JULER, HENRY EDWARD, 23, Cavendish Square, W.

1878 KEETLEY, CHARLES ROBERT BELL, 56, Grosvenor Street, W. (C. 1895–8.) *Trans.* 2.

1898 KELLOCK, THOMAS HERBERT, 8, Queen Anne Street, Cavendish Square, W. *Trans.* 2, *C.C.* 2.

O.M. KELLY, CHARLES, M.D., Ellesmere, Gratwicke Road, Worthing, Sussex.

1882 KESTEVEN, WILLIAM HENRY, Hillwood, Waverley Grove, Hendon, N.W. *Trans.* 2.

1897 KEYSER, CHARLES RALPH, 29, Hamilton Terrace, N.W.

1883 KIDD, PERCY, M.D. (*Hon. Secretary*), 60, Brook Street, W. (C. 1891–2. S. 1899– .) *Trans.* 4, *C.C.* 7.

1887 Knaggs, R. LAWFORD, B.C., 39, Park Square, Leeds. *Trans.* 2.

1878 LACEY, THOMAS WARNER, 196, Burrage Road, Plumstead.

1897 LAMPLOUGH, CHARLES, Chatteris, Cambridgeshire.

1890 LANCASTER, ERNEST LE CRONIER, M.B., B.Ch., Winchester House, Swansea, S. Wales. *Trans.* 2.

1895 LANE, JAMES ERNEST, 46, Queen Anne Street, W. *Trans.* 1.

1883 LANE, WILLIAM ARBUTHNOT, M.B., M.S., 21, Cavendish Square, W. (C. 1893–6.) *Trans.* 29, *C.C.* 10.

O.M. LANGTON, JOHN, 62, Harley Street, W. (C. 1878–80, V.P. 1892–4, P. 1897–9.) *Trans.* 5.

1886 LANKESTER, HERBERT, M.D., Ingleborough, Woking, Surrey.

1885 LARDER, HERBERT, Whitechapel Infirmary, Vallance Road, N.E. *C.C.* 1.

1893 LAWSON, ARNOLD, M.D., 12, Harley Street, W.

O.M. LAWSON, GEORGE, 12, Harley Street, W. (S. 1871–3, C. 1874–6, V.P. 1881–3.) *Trans.* 16.

1900 LEAF, CECIL H., M.B., 75, Wimpole Street, Cavendish Square, W.

1896 Leech, PRIESTLEY, M.D., King Cross, Halifax.

1877 Lediard, HENRY AMBROSE, M.D., 35, Lowther Street, Carlisle. (C. 1889.) *Trans.* 5, *C.C.* 1.

1877 LEES, DAVID B., M.D., 22, Weymouth Street, W. (C. 1885.) *Trans.* 6.

1893 LENDON, EDWIN HARDING, M.B., 162, Holland Park Avenue, W.

ELECTED

1899 LESLIE, MURRAY, M.D., 53, Queen Anne Street, Cavendish Square, W.

1901 LEWERS, ARTHUR HAMILTON NICHOLSON, M.D., 72, Harley Street, Cavendish Square, W.

1892 LEWIS, EDWARD JOHN, M.B., B.C., 74, Hamilton Terrace, N.W.

1895 LEWIS, ERNEST E., M.D., Springfield, Chelmsford.

1879 LICHTENBERG, GEORGE, M.D., 47, Finsbury Square, E.C.

1890 LITTLE, JOHN FLETCHER, M.B., 32, Harley Street, W. *C.C.* 5.

1868 LITTLE, LOUIS STROMEYER, Shanghai, China.

1891 LITTLEWOOD, HARRY, 40, Park Square, Leeds. *Trans.* 1.

1875 LIVEING, EDWARD, M.D., 52, Queen Anne Street, W.

1885 LOCKWOOD, CHARLES BARRETT, 19, Upper Berkeley Street, W. (C. 1896–9.) *Trans.* 6.

1876 LONGHURST, ARTHUR EDWIN TEMPLE, M.D., 26, Lower Belgrave Street, Eaton Square, S.W. (C. 1889–91.) *Trans.* 1.

1897 LOW, HAROLD, M.B., 10, Evelyn Gardens, South Kensington, S.W.

1899 LOW, V. WARREN, M.D., B.S., 27, Queen Anne Street, Cavendish Square, W.

1881 LUBBOCK, MONTAGU, M.D., 19, Grosvenor Street, W.

1876 LUCAS, R. CLEMENT, M.B., B.S., 50, Wimpole Street, W. (C. 1883–5, V.P. 1901– .) *Trans.* 11. *C.C.* 1.

1894 LUFF, ARTHUR PEARSON, M.D., 31, Weymouth Street, W.

1879 LUNN, JOHN REUBEN, New Marylebone Infirmary, Rackham Street, Ladbroke Grove Road, W. (C. 1890–1.) *Trans.* 8, *C.C.* 13.

1893 LYS, HENRY GRABHAM, M.D., Southbrook, Suffolk Road, Bournemouth.

1889 MACBRIDE, P., M.D., 16, Chester Street, Edinburgh.

1891 MACDONALD, GREVILLE, M.D., 85, Harley Street, W.

1901 MCGAVIN, LAURIE HUGH, 21, Montagu Street, Portman Square, W.

1881 MCHARDY, MALCOLM MACDONALD, 5, Savile Row, W. *Trans.* 1.

1882 MACKENZIE, FREDERIC MORELL, 29, Hans Place, S.W.

1891 MACKENZIE, HECTOR W. G., M.A., M.D., 34, Upper Brook Street, W. *Trans.* 6, *C.C.* 1.

1879 MACKENZIE, STEPHEN, M.D., 18, Cavendish Square, W. (C. 1884, 1888–90, S. 1885–7. V.P. 1900– .) *Trans.* 9, *C.C.* 10.

1884 MACKERN, JOHN, M.B., St. German's Lodge, Shooter's Hill Road, Blackheath, S.E.

1879 MACLAGAN, THOMAS JOHN, M.D., 9, Cadogan Place, S.W. (C. 1889–91.) *Trans.* 3.

1885 MACLAREN, RODERICK, M.D., Portland Square, Carlisle. *Trans.* 1.

1896 MAC RAE, FARQUHAR, M.B., 27A, Lowndes Street, Belgrave Square, S.W.

1898 MACREADY, JONATHAN F. C. H., 42, Devonshire Street, Portland Place, W.

1879 MAGILL, JAMES, M.D., M.C., Coldstream Guards, Queen Anne's Mansions, S.W.

ELECTED

1885 MAGUIRE, ROBERT, M.D., 4, Seymour Street, W. *Trans.* 1.

1881 MAKINS, GEORGE HENRY, C.B. 47, Charles Street, Berkeley Square, W. (C. 1892–4, 1898–1901, S. 1895–8.) *Trans.* 4, *C.C.* 7.

1887 MALCOLM, JOHN D., M.B., C.M., 13, Portman Street, W. (C. 1900– .) *Trans.* 1, *C.C.* 1.

1890 MANSON, PATRICK, M.D., C.M., F.R.S., 21, Queen Anne Street, W. (C. 1895–7.)

1888 Marriott, HYDE, M.B., Dial House, Stockport.

1868 MARSH, F. HOWARD (*President*), 30, Bruton Street, W. (C. 1876–7, 1881–3, S. 1878–80, V.P. 1887–8, T. 1899–1901.) *Trans.* 17, *C.C.* 6.

1887 MARTIN, SIDNEY, M.D., B.S., F.R.S., 10, Mansfield Street, W. (C. 1896–9.)

1888 MASON, DAVID JAMES, M.D., C.M., Rosemont, Maidenhead.

1892 MASTERS, JOHN ALFRED, M.D., 31, Albert Gate, Hyde Park, S.W.

1884 MAUDSLEY, HENRY CARR, M.D., 22, Collins Street, Melbourne, Victoria.

1897 MAY, CHICHESTER GOULD, M.D., 59, Cadogan Place, S.W.

1868 May, EDWARD HOOPER, M.D., High Cross, Tottenham, Middlesex.

1888 MAY, WILLIAM PAGE, M.D., B.Sc., 49, Welbeck Street, W.; and Helouan, nr. Cairo, Egypt (October to April).

1901 MENZIES, F. U., Edinburgh University Union, Edinburgh.

1888 MENZIES, J. HERBERT, 47, Earl's Court Square, S.W.

1893 MERCER, WILLIAM BRACEWELL, M.B., B.C., Cliffe Cottage, Ripponden, near Halifax, Yorks.

1878 MEREDITH, WILLIAM APPLETON, C.M., 21, Manchester Square, W. (C. 1887–9.) *Trans.* 2.

1894 MICHELS, ERNST, M.D., 48, Finsbury Square, E.C.

1873 MICKLE, WILLIAM JULIUS, M.D., Grove Hall Asylum, Bow, E. (C. 1897–1900.)

1890 MILEY, MILES, M.A., M.B., 21, Belsize Avenue, Hampstead, N.W.

1896 MILLS-ROBERTS, ROBERT HERBERT, Llanberis, North Wales. *Trans.* 1.

1882 MONEY, ANGEL, M.D., Hunter Street, Sydney, New South Wales. (C. 1888–90.) *Trans.* 3.

1874 MORGAN, JOHN HAMMOND, 68, Grosvenor Street, W. (C. 1883–5. V.P. 1898–1901.) *Trans.* 2, *C.C.* 8.

1888 MORISON, ALEXANDER, M.D., 14, Upper Berkeley Street, W. *Trans.* 2, *C.C.* 1.

1899 MORISON, RUTHERFORD, 14, Saville Row, Newcastle-on-Tyne. *Trans.* 1.

1877 MORRIS, HENRY, M.A., M.B., 8, Cavendish Square, W. (C. 1884–6.) *Trans.* 9.

1877 MORRIS, MALCOLM ALEX., 8, Harley Street, W. (C. 1890–2.) *Trans.* 1, *C.C.* 1.

1901 MORSE, THOMAS HENRY, All Saints' Green, Norwich.

1885 MOTT, FREDERICK WALKER, M.D., F.R.S., 25, Nottingham Place, W. (C. 1895–8.) *Trans.* 1, *C.C.* 4.

ELECTED

1899 MOUAT-BIGGS, CHARLES E. FORBES, 2, Fauconberg Villas, Bayshill, Cheltenham. *C.C.* 2.

1879 MOULLIN, CHARLES W. MANSELL, 69, Wimpole Street, W. (C. 1888–90.) *Trans.* 9, *C.C.* 4.

1899 MOYNIHAN, BERKELEY GEORGE ANDREW, M.S., 33, Park Square, Leeds.

1875 MURPHY, SHIRLEY F., 22, Endsleigh Street, Tavistock Square, W.C. (C. 1888–90.) *C.C.* 1.

1885 MURRAY, ALEXANDER DALTON, M.B., Colombo, Ceylon.

1893 MURRAY, GEORGE REDMAYNE, M.D., 11, Ellison Place, Newcastle-on-Tyne.

1883 MURRAY, HUBERT MONTAGUE, M.D., 25, Manchester Square, W. (C. 1893–6.) *Trans.* 1.

1894 MURRAY, JOHN, 110, Harley Street, W. *C.C.* 1.

1872 MYRTLE, ANDREW S., M.D., 8, Park Parade, Harrogate. (C. 1892.)

1892 NASH, WALTER GIFFORD, 31, St. Peter's, Bedford.

1889 Newman, D., M.D., 18, Woodside Place, Glasgow. *Trans.* 4.

O.M. NORTON, ARTHUR TREHERN, Leyfields Wood, Ashampstead, Berks. (C. 1874–6.) *Trans.* 7.

O.M. NUNN, THOMAS WILLIAM, 8, Stratford Place, W. (C. 1873–4.) *Trans.* 9.

1880 O'CONNOR, BERNARD, M.D., 25, Hamilton Road, Ealing, W. *Trans.* 1.

1899 OGLE, CYRIL, M.B., 96, Gloucester Place, Portman Square, W. *Trans.* 1.

O.M. OGLE, JOHN WILLIAM, M.D., 96, Gloucester Place, Portman Square, W. (C. 1867–8, V.P. 1884–5.) *Trans.* 6.

1868 Ogle, WILLIAM, M.D., The Elms, Driffield Road, Derby.

1887 Oliver, THOMAS, M.D., 7, Ellison Place, Newcastle-upon-Tyne.

1887 OPENSHAW, THOMAS HORROCKS, M.B., 16, Wimpole Street, W. *C.C.* 1.

1863 OPPERT, FRANZ, M.D., 128, Leipzigerstrasse, Friedenau, Germany. *Trans.* 1.

1877 ORD, WILLIAM MILLER, M.D., 37, Upper Brook Street, W., and The Dean, Hurstbourne Tarrant, Andover, Hants. (C. 1882–4, T. 1889–99.) *Trans.* 9, *C.C.* 1.

1889 ORD, W. WALLIS, M.D., B.Ch., The Hall, Salisbury. *Trans.* 1, *C.C.* 1.

1887 ORMEROD, JOSEPH ARDERNE, M.D., 25, Upper Wimpole Street, W. (C. 1899– .)

1884 ORMSBY, LAMBERT HEPENSTAL, M.D., 92, Merrion Square West, Dublin.

1883 ORTON, GEORGE HUNT, M.B., 1A, Campden Hill Road, Kensington, W.

1877 OWEN, ISAMBARD, M.D., 40, Curzon Street, W. (C. 1888–90.) *Trans.* 2.

1888 OXLEY, ALFRED RICE, M.D., Conisbro, St. Julian's Road, Leigham Court Road, Streatham Common, S.W.

1888 PAGE, FREDERICK, M.D., 1, Saville Place, Newcastle-on-Tyne.

1875 PAGE, HERBERT W., M.C., M.B., 146, Harley Street, W. (C. 1882–4, V.P. 1899– .) *Trans.* 7, *C.C.* 2.

1884 PAGET, STEPHEN, 70, Harley Street, W. (C. 1898–1901.) *Trans.* 6, *C.C.* 2.

ELECTED

1873 PARKER, ROBERT WILLIAM, Caryll Hurst, East Grinstead, Sussex. (C. 1882–4, 1890–2, S. 1887–9, V.P. 1893–6.) *Trans.* 15, *C.C.* 7.

1881 PARKER, RUSHTON, M.B., B.S., 59, Rodney Street, Liverpool. (C. 1896–9.) *Trans.* 1, *C.C.* 2.

1890 PARKIN, ALFRED, M.S., 24, Albion Street, Hull. *Trans.* 2.

1894 PARKINSON, JOHN PORTER, M.D., 57, Wimpole street, W. *C.C.* 3.

1898 PARTRIDGE, ARTHUR A. H., M.B., M.Ch., St. Germans, Sutton, Surrey.

1881 PASTEUR, WILLIAM, M.D., 4, Chandos Street, W. (C. 1891–3.) *Trans.* 5, *C.C.* 4.

1893 PATERSON, DONALD ROSE, M.D., C.M., 18, Windsor Place, Cardiff.

1900 PATON, EDWARD PERCY, M.D., 84, Park Street, Grosvenor Square, W.

1892 PAUL, FRANK THOMAS, 38, Rodney Street, Liverpool. *Trans.* 1.

1893 PAUL, JOHN ERNEST, M.B., 26, Queensborough Terrace, Kensington Gardens, W. *Trans.* 1.

1883 PAUL, JOHN LISTON, M.D., 70, Queensborough Terrace, Kensington Gardens, W.

O.M. PAVY, FREDERICK WILLIAM, M.D., F.R.S., 35, Grosvenor Street, W. (C. 1869–71, V.P. 1882–4.) *Trans.* 3.

1886 PAYNE, JOSEPH FRANK, M.D., 78, Wimpole Street, W. (C. 1897–1900.) *Trans.* 1.

1879 PEEL, ROBERT, 120, Collins Street East, Melbourne, Victoria.

1886 Penny, WILLIAM JOHN, Coombe, West Crewkerne, Somerset.

1887 PENROSE, FRANCIS GEORGE, M.D., 84, Wimpole Street, W. (C. 1896–9.) *Trans.* 1, *C.C.* 1.

1882 PEPPER, AUGUSTUS JOSEPH, M.S., M.B., 13, Wimpole Street, W. *Trans.* 1.

1900 PERKINS, GEORGE STEELE, M.D., 85, Wimpole Street, W.

1898 PERKINS, J. J., M.D., 41, Wimpole Street, Cavendish Square, W. *Trans.* 1, *C.C.* 1.

1895 PHEAR, ARTHUR G., M.D., B.C., 47, Weymouth Street, Portland Place, W. *Trans.* 2.

1895 PHILLIPS, F. B. WILLMER, M.B., 7, Harper Place, Bedford.

1884 PHILLIPS, SIDNEY, M.D., 62, Upper Berkeley Street, W. (C. 1893–6.) *Trans.* 7, *C.C.* 6.

O.M. PICK, THOMAS PICKERING, 18, Portman Street, W. (S. 1874–7, C. 1878–80, V.P. 1885–6.) *Trans.* 4.

1885 PITT, GEORGE NEWTON, M.D., 15, Portland Place, W. (C. 1894–6.) *Trans.* 6, *C.C.* 5.

1883 PITTS, BERNARD, M.A., M.C., 109, Harley Street, W. (C. 1893.) *Trans.* 5.

1871 Playne, ALFRED, M.B., Maidenhead.

1884 POLAND, JOHN, 2, Mansfield Street, Cavendish Square, W.

1884 POLLARD, BILTON, B.S., 24, Harley Street, W. (C. 1895–8.) *Trans.* 2.

1868 POLLOCK, JAMES EDWARD, M.D., 52, Upper Brook Street, W. (C. 1878–80.)

ELECTED

1871 POORE, GEORGE VIVIAN, M.D., 24A, Portland Place, W. (C. 1879–81, V. P. 1901– .) *Trans.* 4.

1881 POWELL, H. A., M.A., 44, Sandgate Road, Folkestone.

O.M. POWELL, Sir R. DOUGLAS, Bart., K.C.V.O., M.D., 62, Wimpole Street, W. (C. 1874–6, V.P. 1889–90, P. 1899-1901.) *Trans.* 4.

1868 PRENTIS, CHARLES, Surgeon-Major, Bengal Medical Service; India.

1884 PRINGLE, JOHN JAMES, M.B., 23, Lower Seymour Street, W. (C. 1890–2, 1897–1900.) *Trans.* 1, *C.C.* 3.

1884 PYE-SMITH, PHILIP HENRY, M.D., F.R.S., 48, Brook Street, W. (C. 1890–2.) *Trans.* 1.

1896 PYE-SMITH, RUTHERFORD JOHN, 350, Glossop Road, Sheffield.

1893 RAKE, ALFRED THEODORE, M.B., B.S., 8, Sheriff Road, West Hampstead, N.W.

1895 RAMSAY, HERBERT MURRAY, 35A, Hertford Street, Mayfair, W.

1889 RANKING, JOHN E., M.D., Hanover House, Tunbridge Wells.

1883 READ, THOMAS LAURENCE, 11, Petersham Terrace, Queen's Gate, S.W.

1901 REISSMANN, CHARLES, M.B., 7, Upper Montague Street, Russell Square, W.C.

1889 Rennie, GEORGE EDWARD, M.D., College Street, Hyde Park, Sydney, N.S.W.

1868 RICE, MICHAEL W., M.D., Elmbank, Hargrave, Northamptonshire, (C. 1876–8.)

1898 RICHARDS, JOSEPH STEWART, M.D., B.S., The Infirmary, East Dulwich, S.E. *C.C.* 2.

O.M. RINGER, SYDNEY, M.D., F.R.S., 15, Cavendish Place, W. (C. 1871–2.) *Trans.* 1.

1896 RISDON, WILLIAM ELLIOT, M.D., 81, Chancery Lane, W.C.

1873 Roberts, DAVID LLOYD, M.D., 11, St. John Street, Manchester.

1888 ROBERTS, FRANK ERNEST, Tulse Dale Villa, Lower Norwood, S.E.

1883 ROBERTS, FREDERICK THOMAS, M.D., 102, Harley Street, W. (C. 1892–4.)

1900 ROBERTS, GRIFFITH LLOYD, M.B., C.M., Castle Square, Carnarvon, North Wales.

1890 ROBERTSON, ROBERT, M.D., Belgrave Road, Ventnor, Isle of Wight.

1885 ROBINSON, ARTHUR HENRY, M.D., St. Mary's Infirmary, Highgate Hill, N. *C.C.* 3.

1890 ROBINSON, GEORGE SOMERVILLE, Surgeon-Major, 13, Lupus Street, St. George's Square, S.W.

1892 ROBINSON, HENRY BETHAM, M.D., M.S., 1, Upper Wimpole Street, W. (C. 1901–). *Trans.* 6, *C.C.* 8.

1885 ROBSON, A. W. MAYO, 7, Park Square, Leeds. (C. 1893–6.) *Trans.* 13.

1897 ROGER-SMITH, HUGH, M.D., 1, College Terrace, Fitzjohn's Avenue, N.W. *Trans.* 1.

1889 ROLLESTON, HUMPHRY DAVY, M.A., M.D., 55, Upper Brook Street, Grosvenor Square, W. (C. 1899– .) *Trans.* 9. *C.C.* 5.

1888 ROPER, ARTHUR, "Colby," Lewisham Hill, Blackheath. *Trans.* 1.

1897 ROSS, FREDERICK WILLIAM FORBES, M.D., 15, Gower Street, W.C.

ELECTED

1877 ROTH, BERNARD, 38, Harley Street, Cavendish Square, and "Wayside," Preston Park Avenue, Brighton. *Trans.* 1, *C.C.* 4.

1890 ROUGHTON, EDMUND WILKINSON, B.S., 38, Queen Anne Street, W. *C.C.* 1.

1874 ROWLAND, EDWARD ROGER, Dordrecht, Wodehouse, S. Africa.

1898 RUSSELL, ALFRED ERNEST, M.D., B.S., Melton House, Manor Park, Lee, S.E.

1900 RUSSELL, J. S. RISIEN, M.D., 4, Queen Anne Street, Cavendish Square, W. *Trans.* 1, *C.C.* 1.

1887 RUTHERFOORD, H. T., M.B., Salisbury House, Taunton.

1885 RYLE, REGINALD JOHN, M.D., Green View, Hadley Green, Barnet.

1882 SAINSBURY, HARRINGTON, M.D., 52, Wimpole Street, W. (C. 1898–1901.)

1899 SANDIFER, HENRY STEPHEN, M.D., B.S., 11, Porchester Gardens, W.

1893 SANSOM, ARTHUR ERNEST, M.D., 84, Harley Street, W. *Trans.* 1, *C.C.* 1.

1873 SAVAGE, GEORGE HENRY, M.D., 3, Henrietta Street, W. (C. 1882–3.)

1886 SAVILL, THOMAS DIXON, M.D., 60, Upper Berkeley Street, W. *Trans.* 1, *C.C.* 3.

1899 SCHORSTEIN, GUSTAVE, M.B., 11, Portland Place, W.

1886 SCOTT, ALFRED, 15, German Place, Brighton.

1894 SCOTT, BERNARD, "Hartington," Poole Road, Bournemouth.

1900 SCOTT, KENNETH, M.B., 7, Manchester Square, W.

1892 SCOTT, RICHARD JAMES HERBERT, 28, Circus, Bath. *C.C.* 1.

1869 SEDGWICK, LEONARD WILLIAM, M.D., 48, Gloucester Terrace, Hyde Park, W. (C. 1879–81, V.P. 1897–1900.)

1897 SEGUNDO, CHARLES SEMPILL DE, M.B., B.S., 6, Brook Street, Hanover Square, W.

1892 SELWYN-HARVEY, JOHN STEPHENSON, M.D., 1, Astwood Road, S.W.

1878 SEMON, Sir FELIX, M.D., 39, Wimpole Street, W. (C. 1885–7.) *Trans.* 8, *C.C.* 3.

1884 SHARKEY, SEYMOUR J., M.D., 22, Harley Street, W. (C. 1895–8.)

1898 SHAW, HERBERT BATTY, M.D., 7, Devonshire Place, Portland Place, W. *C.C.* 3.

1889 SHAW, LAURISTON ELGIE, M.D., 64, Harley Street, W. *C.C.* 1.

1875 SHERWOOD, ARTHUR PAUL, 8, Seaside Road, Eastbourne.

1886 SILCOCK, ARTHUR QUARRY, M.D., B.S., 52, Harley Street, W. (C. 1896–9.) *Trans.* 5. *C.C.* 2.

1879 SKERRITT, EDWARD MARKHAM, M.D., Edgecumbe House, Richmond Hill, Clifton, Bristol. (C. 1895–8.) *Trans.* 2.

1872 SLIGHT, GEORGE, M.D., 14, Old Burlington Street, W.

1882 SMITH, E. NOBLE, 24, Queen Anne Street, W. *Trans.* 1.

1896 SMITH, EBENEZER STANLEY, M.D., 68, Wimpole Street, Cavendish Square, W.

1888 SMITH, FREDERICK J., M.D., 138, Harley Street, W. *Trans.* 2.

ELECTED

1884 SMITH, R. PERCY, M.D., 36, Queen Anne Street, Cavendish Square, W. (C. 1898–1901.)

1893 SMITH, SOLOMON CHARLES, M.D., Four Oaks, Walton-on-Thames.

O.M. SMITH, Sir THOMAS, Bart., 5, Stratford Place, W. (C. 1869–71, V.P. 1880–2.) *Trans.* 14.

1894 SMITH, THOMAS RUDOLPH, M.B., B.C., 25, Bridge Road, Stockton-on-Tees.

1893 SNAPE, ERNEST ALFRED, M.D., 39, Welbeck Street, W.

1868 SNOW, WILLIAM V., M.D., Richmond Gardens, Bournemouth.

1890 SOLLY, ERNEST, M.B., Strathlea, Harrogate, Yorks. *C.C.* 1.

1888 SPENCER, WALTER GEORGE, M.S., M.B., 35, Brook Street, W. (C. 1896–9.) *Trans.* 16, *C.C.* 9.

1885 SPICER, FREDERICK, M.D., 57, Devonshire Street, W.

1888 SPICER, ROBERT HENRY SCANES, M.D., 28, Welbeck Street, W. *C.C.* 1.

1882 SPOONER, FREDERICK HENRY, M.D., 4, Maitland Place, Lower Clapton, N.E.

1896 SPURRELL, CHARLES, Medical Superintendent, Poplar and Stepney Sick Asylum, Devon's Road, Bromley-by-Bow, E.

1876 SQUIRE, A. BALMANNO, 24, Weymouth Street, W. *Trans.* 5, *C.C.* 4.

1892 STABB, EWEN CARTHEW, 57, Queen Anne Street, W. *C.C.* 1.

1879 STAPLES, FRANCIS PATRICK, Brigade-Surgeon, Army (retired). (Address uncommunicated.)

1896 STEWARD, FRANCIS JAMES, M.B., B.S., 24, St. Thomas's Street, London Bridge, S.E. *Trans.* 2.

1874 Stirling, EDWARD C., M.D. [care of Messrs. ELDER & Co., 7, St. Helen's Place, E.C.], Adelaide, South Australia.

1888 STOKER, GEORGE, 14, Hertford Street, W. *C.C.* 2.

1899 STONE, WILLIAM GREAM, M.B., 93, Denmark Hill, S.E.

1884 STONHAM, CHARLES, 4, Harley Street, W. *C.C.* 3.

1878 STRUGNELL, FREDERICK WILLIAM, Grove End House, Highgate Road, Highgate, N.W. *C.C.* 1.

1878 Sturge, WILLIAM ALLEN, M.D., 29, Boulevard Dubouchage, Nice, France.

1894 SUTHERLAND, GEORGE A., M.D., 73, Wimpole Street, Cavendish Square, W. *Trans.* 1.

1872 SUTHERLAND, HENRY, M.D., 21, New Cavendish Street, Portland Place, W. *Trans.* 1.

1882 SYMONDS, CHARTERS JAMES, M.S., 58, Portland Place, W. (C. 1888–9, 1901– , S. 1898–1901.) *Trans.* 12, *C.C.* 14.

1876 SYMONDS, HORATIO PERCY, 35, Beaumont Street, Oxford.

1885 TAIT, EDWARD SABINE, M.D., 48, Highbury Park, N.

1885 TAIT, HENRY BREWER, Lincluden, Sunnyside Road, Hornsey Lane, N.

1891 TATE, WALTER WILLIAM HUNT, M.D., 32, Queen Anne Street, W.

1886 TAY, WAREN, 4, Finsbury Square, E.C.

1878 TAYLER, FRANCIS THOMAS, M.B., 224, Lewisham High Road, S.E.

ELECTED

1897 TAYLOR, E. CLAUDE, M.D., B.S., Eland House, Rosslyn Hill, Hampstead, N.W.

1875 TAYLOR, FREDERICK, M.D., (*Treasurer*), 20, Wimpole Street, W. (S. 1879–81, C. 1882–4, V.P. 1894–7.) *Trans.* 13, *C.C.* 3.

1889 Taylor, HENRY HERBERT, 10, Brunswick Place, Brighton.

1890 TAYLOR, JAMES, M.D., 49, Welbeck Street, W. *C.C.* 3.

1882 TAYLOR, SEYMOUR, M.D., 16, Seymour Street, W. *Trans.* 1, *C.C.* 1.

1885 Taylor, W. C. EVERLEY, 34, Queen Street, Scarborough.

1886 TEALE, THOMAS PRIDGIN, M.B., F.R.S., 38, Cookridge Street, Leeds. (C. 1897–1900.)

1896 TEMPLETON, GEORGE, M.B., 10, Seymour Street, Portman Square, W.

1890 THANE, EDGAR HERBERT, M.D., Wagga-Wagga, New South Wales.

1882 THIN, GEORGE, M.D., 63, Harley Street, W. *Trans.* 1.

1886 THOMPSON, CHARLES HERBERT, M.D., 17, New Cavendish Street, Portland Place, W.

O.M. THOMPSON, EDMUND SYMES, M.D., 33, Cavendish Square, W. (C. 1880–2.) *Trans.* 1.

O.M. THOMPSON, Sir HENRY, Bart., 35, Wimpole Street, W. (C. 1867–8, V.P. 1886–7.) *Trans.* 3.

1897 THOMSON, H. CAMPBELL, M.D., 34, Queen Anne Street, Cavendish Square, W. *C.C.* 1.

1894 THOMSON, STCLAIR, M.D., 28, Queen Anne Street, W. *Trans.* 2, *C.C.* 7.

1896 THORNE, WILLIAM BEZLY, 53, Upper Brook Street, W. *C.C.* 2.

1887 THORNTON, JOHN KNOWSLEY, M.B., C.M., Hildersham Hall, Cambridge. (C. 1890–1.)

1872 THORNTON, WILLIAM PUGIN, 35, St. George's Road, Canterbury, *Trans.* 5.

1885 THURSFIELD, THOMAS WILLIAM, M.D., Selwood, Beauchamp Square, Leamington.

1899 TILLEY, HERBERT, M.D., 89, Harley Street, Cavendish Square, W. *C.C.* 1.

1891 TOMSON, W. BOLTON, M.D., Park Street West, Luton, Bedfordshire.

1897 TOOGOOD, F. SHERMAN, M.D., The Infirmary, 282, High Street, Lewisham, S.E.

1892 TOOTH, HOWARD HENRY, C.M.G., M.D., 34, Harley Street, W. *Trans.* 1.

1887 TOTSUKA, KANKAI, Tokio, Japan.

1874 TRAVERS, WILLIAM, M.D., 2, Phillimore Gardens, Kensington, W.

1884 TREVES, Sir FREDERICK, C.B., K.C.V.O., 6, Wimpole Street, W. (C. 1893.) *Trans.* 7.

1897 TUBBY, ALFRED HERBERT, M.S., 25, Weymouth Street, Portland Place, W. *Trans.* 2, *C.C.* 2.

1899 TULLIS, GEORGE AUGUSTUS, M.D., Holmbush, Grove Road, Southsea.

1882 TURNER, GEORGE ROBERTSON, 41, Half Moon Street, W. (C. 1897–1900.) *Trans.* 8. *C.C.* 4.

1888 TURNER, PHILIP DYMOCK, M.D., Sudbury, Isle of Wight. *Trans.* 1.

1898 TURNER, WILLIAM, M.B., 53, Queen Anne Street, Cavendish Square, W.

ELECTED

1893 TURNEY, HORACE GEORGE, M.D., 68, Portland Place, W. *C.C.* 4.

1878 TYSON, WILLIAM JOSEPH, M.D., 10, Langhorne Gardens, Folkestone. (C. 1886–8, V.-P. 1900– .) *Trans.* 7.

1881 UHTHOFF, JOHN CALDWELL, M.D., Wavertree House, Brunswick Place, Brighton. (C. 1898–1901.)

1868 VENNING, EDGCOMBE, 30, Cadogan Place, S.W. (C. 1876–8.) *Trans.* 2.

1890 VOELCKER, ARTHUR FRANCIS, M.D., B.S., 31, Harley Street, W. (C.1900– .) *Trans.* 2, *C.C.* 2.

1886 WADE, CHARLES H., 19, Royal Terrace, Southend-on-Sea.

1868 WAGSTAFFE, WILLIAM WARWICK, Purleigh, St. John's Hill, Sevenoaks. (C. 1878.)

1886 **WAINEWRIGHT**, BENJAMIN, M.B., C.M., 47, Weymouth Street, W. *Trans.* 2, *C.C.* 2.

1885 WAKLEY, THOMAS, jun., 5, Queen's Gate, S.W.

1885 WALKER, CHARLES ROTHERHAM, M.D., 15, Sackville Road, Hove, Sussex.

1899 WALKER, HENRY ROE, 20, St. James's Place, S.W.

1896 WALLACE, CUTHBERT SYDNEY, St. Thomas's Hospital, S.E. *Trans.* 3, *C.C.* 2.

1890 WALLIS, FREDERICK CHARLES, M.B., B.C., 26, Welbeck Street, W. *Trans.* 3, *C.C.* 7.

1875 WALSHAM, WILLIAM J., C.M., 77, Harley Street, W. (C. 1882–4, V.P. 1896–9.) *Trans.* 5, *C.C.* 1.

1888 WALTERS, FREDERICK RUFENACHT, M.D., 21, Wimpole Street, W. *C.C.* 2.

1896 WARD, EDWARD, M.B., 22, Park Place, Leeds. *Trans.* 1.

1888 Warner, PERCY, Rydal, Woodford Green, Essex.

1894 WASHBOURN, JOHN WICHENFORD, M.D., 6, Cavendish Place, Cavendish Square, W. *Trans.* 3, *C.C.* 1.

1891 WATERHOUSE, HERBERT FURNIVALL, M.D., C.M., 81, Wimpole Street, W.

1896 **WEBER**, FRED. PARKES, M.D., 19, Harley Street, W. *C.C.* 5.

O.M. WEBER, Sir HERMANN D., M.D., 10, Grosvenor Street, W. (C. 1867–71, V.P. 1873–5.) *Trans.* 9.

1882 WEST, SAMUEL, M.D., 15, Wimpole Street, W. (C. 1887–9.) *Trans.* 16, *C.C.* 5.

1895 WETHERED, FRANK J., M.D., 83, Harley Street, W.

1874 WHEELHOUSE, CLAUDIUS GALEN, Hilary Place, Leeds. *Trans.* 1.

1868 WHIPHAM, THOMAS TILLYER, M.D., 11, Grosvenor Street, W. (C. 1878–80, V.P. 1894–7.) *Trans.* 13.

1891 WHITE, CHARLES PERCIVAL, M.B., B.C., 22, Cadogan Gardens, S.W.

1882 WHITE, EDWIN FRANCIS, Westlands, 280, Upper Richmond Road, Putney, S.W.

1890 WHITE, GILBERT B. MOWER, M.B., B.S., 112, Harley Street, W.

1883 WHITE, WILLIAM HALE, M.D. 65, Harley Street, W. (C. 1887–9, 1896–9, S. June, 1893–6.) *Trans.* 14, *C.C.* 4.

1883 WHITE, WILLIAM HENRY, M.D., 43, Weymouth Street, W. *Trans.* 1. *C C.* 1.

ELECTED

1894 WHITELOCKE, RICHARD HENRY ANGLIN, M.B., C.M., 6, Banbury Road, Oxford.

1897 WHITFIELD, ARTHUR, M.D., 12, Upper Berkeley Street, Portman Square, W.

1897 WHITING, ARTHUR JOHN, M.D., 142, Harley Street, W.

1882 WHITTLE, EDWARD GEORGE, M.D., 65, Dyke Road, Brighton.

1871 WIGHT, GEORGE, M.B., C.M., 428, Liverpool Road, N.

1879 WILCOX, HENRY, M.B., Newlyn, Fleet, Hants.

1894 WILKIN, GRIFFITH CHARLES, West Coker, near Yeovil, Somerset.

1884 WILLCOCKS, FREDERICK, M.D., 14, Mandeville Place, W. *C.C.* 1.

O.M. WILLETT, ALFRED, 36, Wimpole Street, W. (C. 1872–5, V.P. 1889–91.) *C.C.* 2.

1890 WILLETT, EDGAR, M.B., 25, Welbeck Street, W.

1898 WILLEY, FREDERICK INGOR, M.B., B.S., The Wych, 8, Avenue Road, Highgate, N.

1888 WILLIAMS, CAMPBELL, 18, Queen Anne Street, Cavendish Square, W. *Trans.* 2.

O.M. WILLIAMS, CHARLES THEODORE, M.D., 2, Upper Brook Street, W. (C. 1877–9, V.P. 1889–90.) *Trans.* 8.

1888 WILLIAMS, DAWSON, M.D., 2, Wyndham Place, Bryanston Square, W. (C. 1893–7.)

1881 WILLIAMS, Sir JOHN, Bart., M.D., 63, Brook Street, W. (C. 1885–6.)

1890 WILLIAMS, W. ROGER, Beaufort House, Clifton Down, Clifton, Bristol.

1876 WILLIAMSON, JAMES MANN, M.D., Ventnor, Isle of Wight.

O.M. WILLIS, FRANCIS, M.D., The Spa, Braceborough, Stamford.

1893 WILLS, JOSEPH PEARCE BUDGETT, M.D., Bexhill, Hastings.

1889 WILLS, WILLIAM ALFRED, M.D., 29, Lower Seymour Street, W.

1886 WILSON, ALBERT, M.D., Minto House, Fairlop Road, Leytonstone, Essex. *Trans.* 1, *C.C.* 2.

1888 Wilson, CLAUDE, M.D., C.M., Belmont, Tunbridge Wells. *Trans.* 2.

1891 WILSON, THEODORE STACEY, M.D., C.M., 65, Temple Row, Birmingham. *Trans.* 1.

1890 WOOD, NEVILLE, 42, Elvaston Place, Queen's Gate, S.W.

1883 WOODCOCK, JOHN ROSTRON, Abberley, near Stourport, Worcestershire.

1879 WOODWARD, GEORGE P. M., M.D., Deputy Surgeon-General; Sydney, New South Wales.

1894 WOOLLETT, CHARLES JEROME, "Ambleside," Streatham, S.W.

1884 WORTS, EDWIN, 6, Trinity Street, Colchester.

1888 WYMAN, WILLIAM S., M.D., Red Brae, 18, Putney Hill, S.W.

1892 WYNTER, WALTER ESSEX, M.D., B.S., 30, Upper Berkeley Street, W. *C.C.* 2.

1872 YEO, I. BURNEY, M.D., 44, Hertford Street, W. (C. 1881–3.) *Trans.* 8.

[*It is requested that any change of Title or Residence be communicated to the Secretaries before the 1st of July in each year, in order that the list may be made as correct as possible.*]

LIST OF MEMBERS.

ORIGINAL MEMBERS (ALPHABETICALLY).

Henry Arnott.
Richard Barwell.
Henry Charlton Bastian, M.D., F.R.S.
Sir Wm. Henry Broadbent, Bart., M.D., F.R.S.
Thomas Bryant.
Thomas Buzzard, M.D.
William Cayley, M.D.
Sir William Selby Church, Bart., M.D.
Edward Clapton, M.D.
John Couper.
William Howship Dickinson, M.D.
Sir Dyce Duckworth, M.D.
Christopher Heath.
Timothy Holmes.
Jonathan Hutchinson, F.R.S.
J. Hughlings Jackson, M.D., F.R.S.
Charles Kelly, M.D.
John Langton.
George Lawson.
Arthur Trehern Norton.
Thomas William Nunn.
John William Ogle, M.D.
Frederick William Pavy, M.D., F.R.S.
Thomas Pickering Pick.
Sir Richard Douglas Powell, Bart., M.D., K.C.V.O.
Sydney Ringer, M.D., F.R.S.
Sir Thomas Smith, Bart.
Edmund Symes Thompson, M.D.
Sir Henry Thompson, Bart.
Sir Hermann D. Weber, M.D.
Alfred Willett.
Charles Theodore Williams, M.D.
Francis Willis, M.D.

ARRANGED ACCORDING TO DATE OF ELECTION.

1868 Constantine Holman, M.D.
Thomas Tillyer Whipham, M.D.
Christian G. H. Bäumler, M.D.
James Grey Glover, M.D.
T. Henry Green, M.D.
Howard Marsh.
Charles Prentis.
Edgcombe Venning.

1868 John Ford Anderson, M.D.
George Granville Bantock, M.D.
George Charles Bright, M.D.
Frank W. Cooper.
Julian Evans, M.B.
Edward Hooper May, M.D.
William Warwick Wagstaffe.
William Ogle, M.D.

1868 James Edward Pollock, M.D.
Franz Oppert, M.D.
William V. Snow, M.D.
Charles Drage, M.D.
Frederick Royston Fairbank, M.D.
Michael W. Rice, M.D.
Louis Stromeyer Little.
1869 Leonard William Sedgwick, M.D.
J. Warrington Haward.
Henry Frederick Augustus Goodridge, M.D.
Olliver Thomas Duke, M.B.
1871 Alfred Playne, M.B.
George Wight, M.B.
Ebenezer Diver, M.D.
George Vivian Poore, M.D.
1872 I. Burney Yeo, M.D.
Henry Harris, M.D.
William Pugin Thornton.
G. Anderson Critchett.
J. C. J. Fenwick, M.D.
Andrew J. Myrtle, M.D.
Sir William Bartlett Dalby.
Thomas Ridge Jones, M.D.
George Slight, M.D.
Henry Sutherland, M.D.
1873 William Julius Mickle, M.D.
Robert William Parker.
David Lloyd Roberts, M.D.
George Henry Savage, M.D.
Edwin Chisholm, M.D.
Thomas Churton, M.D.
1874 John Hammond Morgan.
Edward R. Rowland.
Claudius Galen Wheelhouse.
Edward C. Stirling, M.D.
William Henry Bennett.
William Travers, M.D.
William Brown Holderness.
Andrew Clark.
1875 Sir Thomas Barlow, Bart., K.C.V.O., M.D.
Sidney Coupland, M.D.
Clinton T. Dent.
C. D. B. Hale.
Edward Liveing, M.D.
William J. Walsham, C.M.
Rickman John Godlee, M.S.
Arthur Paul Sherwood.
T. Gilbart-Smith, M.D.
James Frederic Goodhart, M.D.
Sir William Richard Gowers, M.D., F.R.S.
1875 William Smith Greenfield, M.D.
Shirley F. Murphy.
Herbert W. Page.
Frederick Taylor, M.D.
1876 Arthur E. J. Barker.
Horatio Percy Symonds.
A. Balmanno Squire.
David White Finlay, M.D.
Henry Greenway Howse, M.S.
Furneaux Jordan.
R. Clement Lucas, B.S.
James Mann Williamson, M.D.
George Buckston Browne.
Arthur Edwin Temple Longhurst, M.D.
1877 Robert Hogarth Clay, M.D.
A. Pearce Gould, M.S.
Henry Radcliffe Crocker, M.D.
David B. Lees, M.D.
Walter Hamilton Acland Jacobson, M.B., M.Ch.
Isambard Owen, M.D.
William Ewart, M.D.
Henry Morris, M.B.
William Miller Ord, M.D.
Henry Ambrose Lediard, M.D.
Bernard Roth.
Henry Hugh Clutton.
Malcolm Alex. Morris.
1878 George P. Field.
Thomas Warner Lacey.
Thomas Colcott Fox, M.B.
Sir Felix Semon, M.D.
Henry de Fonmartin, M.D.
C. H. Golding-Bird, M.B.
Donald William Charles Hood, M.D.
Lord Lister, F.R.S.
Francis Thomas Tayler, M.B.
F. de Havilland Hall, M.D.
William Allen Sturge, M.D.
William Joseph Tyson, M.D.
William Johnston, M.D.
Charles Robert Bell Keetley.
William Appleton Meredith, C.M.
1879 Frederick William Strugnell.
William Edward Burton.
James Magill, M.D.
Edward Markham Skerritt, M.D.
Henry Wilcox, M.B.
John Abercrombie, M.D.
F. G. Dawtrey Drewitt, M.D.
Stephen Mackenzie, M.D.
William Harrison Cripps.

1879 Francis Patrick Staples.
Geo. Courtenay Henderson, M.D.
Thomas John Maclagan, M.D.
Henry Davy.
Thos. Walter Harropp Garstang.
George Lichtenberg, M.D.
Charles W. Mansell Moullin.
John Reuben Lunn.
George P. M. Woodward, M.D.
Robert Peel.
Frederic S. Dennis, M.D.
1880 T. Mark Hovell.
Wyndham Cottle, M.D.
Henry Francis Baker.
Bernard O'Connor, M.D.
Charles Edward Beevor, M.D.
1881 George Henry Makins, C.B.
Robert William Burnet, M.D.
James Kingston Fowler, M.D.
Charles Edward Harrison, M.B.
Malcolm Macdonald McHardy.
Rushton Parker.
Sir John Williams, Bart, M.D.
Montagu Lubbock, M.D.
William Pasteur, M.D.
John Caldwell Uhthoff, M.D.
Henry Treutham Butlin.
H. A. Powell, M.A.
1882 George Robertson Turner.
E. Noble Smith.
Robert William Goldie.
Frederick Charles Barker, M.D.
William Henry Kesteven.
Frederic Morell Mackenzie.
Daniel Colquhoun, M.D.
Seymour Taylor, M.D.
Philip Henry Bindley, M.B.
Edward George Whittle, M.D.
D. H. Goodsall.
Frederick Henry Spooner, M.D.
Frederick Haycraft Berry, M.D.
Herbert Collier, M.D.
Samuel West, M.D.
Emile Emond, M.D.
Charters James Symonds, M.S.
Angel Money, M.D.
C. F. Coxwell, M.B.
George Allan Heron, M.D.
Augustus Joseph Pepper, M.B.
Harrington Sainsbury, M.D.
George Thin, M.D.
Edwin Francis White.
1883 Anthony A. Bowlby, C.M.G.
Percy Kidd, M.D.
1883 William Henry White, M.D.
Hubert Montague Murray, M.D.
Robert Fitzroy Benham.
William Henry Allchin, M.D.
John Mitchell Bruce, M.D.
William Arbuthnot Lane, M.S.
Bernard Pitts.
William Hale White, M.D.
William Coode Adams, M.B.
Robert Leamon Bowles, M.D.
George Henry Jackson.
George Hunt Orton, M.B.
John Liston Paul, M.D.
Thomas Laurence Read.
Frederick Thomas Roberts, M.D.
Charles Alfred Ballance, M.S.
John Rostron Woodcock.
1884 Frederick Willcocks, M.D.
R. Percy Smith, M.D.
Edgar Duke.
John Mackern, M.B.
Paul M. Chapman, M.D.
Wilmot Parker Herringham, M.D.
Philip Henry Pye-Smith, M.D. F.R.S.
Charles Stonham.
Dudley Wilmot Buxton, M.D.
Edwin Worts.
Seymour J. Sharkey, M.D.
Sir Frederick Treves, C.B., K.C.V.O.
John James Pringle, M.B.
Frederick Lucas Benham, M.D.
Walter Edmunds, M.C.
Stephen Paget.
Lambert Hepenstal Ormsby, M.D.
John Poland.
Edwin Leonard Adeney, M.D.
Victor Horsley, F.R.S.
Henry Carr Maudsley, M.D.
Bilton Pollard, B.S.
1885 Frederick Spicer, M.B.
Herbert Larder.
James Berry, B.S.
Fred. Walker Mott, M.D., F.R.S.
George Newton Pitt, M.D.
W. C. Everley Taylor.
Sidney Phillips, M.D.
A. W. Mayo Robson.
Thomas Wakley, jun.
Herbert William Allingham.
Thomas William Thursfield, M.D.
Alexander Dalton Murray, M.B.
Robert Maguire, M.D.

1885 Robert Alexander Gibbons, M.D.
Henry Brewer Tait.
Charles Rotherham Walker, M.D.
Richard Caton, M.D.
Arthur Henry Robinson, M.D.
Edward Sabine Tait, M.B.
William Bruce Clarke, M.B.
Charles Barrett Lockwood.
Reginald J. Ryle, M.D.
J. Michell Clarke, M.B.
Henry George Armstrong.
Roderick Maclaren, M.D.
W. Watson Cheyne, F.R.S.
Edward Liveing Fenn, M.D.
1886 Thomas Dixon Savill, M.D.
John Cahill.
Charles Henry Wade.
Benjamin Wainewright, M.B.
Waren Tay.
William John Penny.
William Henry Battle.
James Hardie, M.D.
Francis Henry Hawkins, M.D.
R. Hingston Fox, M.D.
Henry Edward Juler.
John Ward Cousins, M.D.
Joseph Frank Payne, M.D.
T. Pridgin Teale, F.R.S.
H. Lankester, M.D.
Arthur T. Davies, M.D.
Charles Herbert Thompson, M.D.
Arthur Quarry Silcock, M.D., B.S.
Henry Handford, M.D.
Alfred Scott.
Albert Wilson, M.D.
1887 Archibald E. Garrod, M.D.
H. T. Rutherfoord, M.B.
Kankai Totsuka.
Thomas Oliver, M.D.
Francis George Penrose, M.D.
Samuel Herbert Habershon, M.D.
John Knowsley Thornton.
John Bland-Sutton.
Oswald Auchinleck Browne, M.D.
Joseph Arderne Ormerod, M.D.
J. H. E. Brock, M.B., B.S.
Francis William Clark.
A. H. Weiss Clemow, M.D., C.M.
E. Hurry Fenwick.
R. Lawford Knaggs, B.C.
John D. Malcolm, M.B., C.M.
Sidney Martin, M.D., B.S., F.R.S.
Thomas Horrocks Openshaw, M.B.
1888 A. G. Barrs, M.D.
1888 J. W. Batterham, M.B., B.S.
Montagu Handfield-Jones, M.D.
Alfred Rice Oxley, M.D.
Arthur Roper, M.D.
Robert Henry Scanes Spicer, M.D.
Campbell Williams.
Frederic S. Eve.
Alexander Morison, M.D.
Frederick Page, M.D.
Frederick J. Smith, M.D.
Frederick R. Walters, M.D.
Claude Wilson, M.D., C.M.
Charles H. Gage-Brown, M.D.
J. H. Menzies.
Frank Ernest Roberts.
George Stoker.
Robert Ashton Bostock.
Hugh Armstrong.
Hyde Marriott, M.B.
Percy Warner.
J. T. James, M.D.
Edwin A. Barton.
W. Page May, M.D.
Philip D. Turner, M.D.
William S. Wyman, M.D.
Dawson Williams, M.D.
Augustus W. Addinsell, M.B., C.M.
John Anderson, M.D.
Henry French Banham, M.D.
George Haynes Hetherington.
David James Mason, M.D., C.M.
Walter G. Spencer, M.B., M.S.
1889 Theodore Dyke Acland, M.D.
Raymond Johnson, M.B., B.S.
H. D. Rolleston, M.A., M.D.
P. MacBride, M.D.
D. Newman, M.D.
Herbert Elwin Harris, M.B.
John E. Ranking, M.D.
William Alfred Wills, M.D.
Edward Ashby Fardon.
Stanley Boyd, M.B.
George Ezra Halstead, M.D., B.S.
Henry Herbert Taylor.
John Duncan, M.D.
Wm. Wallis Ord, M.D., B.Ch.
Leonard Arthur Bidwell.
Arthur J. M. Bentley, M.D.
Francis R. B. Bisshopp, M.B.
Henry Percy Dean, M.B.
Louis Albert Dunn, M.S.
Daniel Mackay Forbes.

1889 H. Pennell Hawkins, M.D., B.Ch.
Lauriston Elgie Shaw, M.D.
1890 John Rose Bradford, M.D., F.R.S.
J. Fletcher Little, M.B.
Robert Robertson, M.D.
Ernest Solly, M.B.
James Taylor, M.D.
Francis O. Buckland, B.A., M.B., C.M.
G. Somerville Robinson.
Edmund W. Roughton, B.S.
Edgar Willett, M.B.
Thomas H. Rickard Crowle.
James Calvert, M.D.
H. Roxburgh Fuller, M.D.
Arthur F. Voelcker, M.D.
Neville Wood.
W. Roger Williams.
Gilbert B. M. White, M.B., B.S.
Frederick Charles Wallis, M.B., B.C.
Alfred Parkin, M.S.
George A. Hawkins-Ambler.
James Harper, M.D.
Walter Henry Brown.
John Walter Carr, M.D.
Ernest Le Cronier Lancaster, M.B., B.Ch.
Patrick Manson, M.D., C.M., F.R.S.
Miles Miley, M.A., M.B.
Edgar Herbert Thane, M.B.
Charles William Chapman, M.D.
Michael G. Foster, M.A., M.B.
1891 Frederic François Burghard, M.D., M.S.
Roger Neville Goodman, M.B.
Herbert Furnivall Waterhouse, M.D., C.M.
Walter William Hunt Tate, M.D.
William Lee Dickinson, M.D.
Greville MacDonald, M.D.
J. Kingston Barton
J. Dundas Grant, M.D.
W. Kington Fyffe, M.B., B.C.
Albert Carless, M.B., M.S.
W. Bolton Tomson, M.D.
Harry Littlewood, M.D.
Hector W. G. Mackenzie, M.A., M.D.
Chas. Percival White, M.B., B.C.
Arnold Caddy.
1891 Theodore Stacey Wilson, M.D., C.M.
1892 William Hunter, M.D.
Frank Thomas Paul.
Walter Essex Wynter, M.D., B.S.
Damer Harrison.
John Alfred Masters, M.D.
Walter Giffard Nash.
John Stephenson Selwyn-Harvey, M.D.
Ewen Carthew Stabb.
Edward John Lewis, M.B., B.C.
Henry Betham Robinson, M.D. M.S.
Richard James Herbert Scott.
Howard Henry Tooth, C.M.G., M.D.
1893 John Ernest Paul, M.B.
James William Bond, M.D.
Harry Campbell, M.D.
W. Soltau Fenwick, M.D.
Ernest Alfred Snape, M.D.
Lewis G. Glover, M.B., B.C.
Solomon Charles Smith, M.D.
William Bracewell Mercer, M.B., B.C.
Robert Henry Cole, M.D.
Donald Rose Paterson, M.D., C.M.
Walter Spencer Anderson Griffith, M.D.
Alfred Theodore Rake, M.B., B.S.
Francis Charles Abbott, M.B., B.S.
James William Browne, M.B.
Edwin Harding Lendon, M.B
George Redmayne Murray, M.D., M.A.
R. J. Bliss Howard, M.D.
Henry Jones Curtis, M.D., B.S.
Henry Albert Caley, M.D.
John Henry Dauber, M.B.
Theodore Henry Ionides, M.B., B.S.
Arthur Foxwell, M.D.
Horace George Turney, M.D.
Henry Grabham Lys, M.D.
Arthur Ernest Sansom, M.D.
John Henry Bryant, M.D.
G. Johnston, M.B.
Willmott H. Evans.
Charles Gibbs.
Robert Stephen Charsley.
Joseph Pearce Budgett Wills, M.D.

1893 Arnold Lawson, M.D.
1894 Herbert Morley Fletcher, M.D.
John Wichenford Washbourn, M.D.
Edward Wilberforce Goodall, M.D.
Harry Gilbert Barling.
Ernst Michels, M.D.
Charles Jerome Woollett.
StClair Thomson, M.D.
George A. Sutherland, M.D.
John Murray.
Richard Henry Anglin Whitelocke, M.B., C.M.
John Attlee, M.D., B.C.
Griffith Charles Wilkin.
Thomas Hugh Dickson, M.B.,B.C.
C. Gordon Brodie.
Bernard Scott.
John Porter Parkinson, M.D.
Thomas Rudolph Smith, M.B., B.C.
Arthur Pearson Luff, M.D.
1895 Herbert Murray Ramsay.
Arthur G. Phear, M.D., B.C.
Ernest E. Lewis, M.D.
Karl Grube, M.D.
Gerald R. Baldwin.
James Galloway, M.D.
F. B. Willmer Phillips, M.B.
Charles Henry Hough.
Leonard George Guthrie, M.D.
James Ernest Lane.
Frank J. Wethered, M.D.
1896 Richard Ackerley.
Charles Spurrell.
Cuthbert Sidney Wallace.
Charles R. Box, M.D.
Walter Stacy Colman, M.D.
Edward Ward, M.B.
Norman Dalton, M.D.
Fred. Parkes Weber, M.D.
Francis James Steward, M.B., B.S.
William Bezly Thorne, M.D.
Douglas Drew, M.D., B.S.
Surg.-Capt. W. R. Crooke-Lawless, A.M.S., M.D.
William Henry Crosse, M.D.
William Elliot Risdon, M.D.
Robert Milne Beaton, M.B.
Priestley Leech, M.D.
Ebenezer Stanley Smith, M.D.
G. H. A. Comyns Berkeley, M.B.
1896 Edward Cooper Bentham.
Robert Herbert Mills-Roberts.
Jonathan Hutchinson, jun.
Frederick E. Batten, M.D.
Farquhar Mac Rae, M.B.
Rutherford John Pye-Smith.
George Templeton.
Bertram Louis Abrahams, M.B., B.Sc.
1897 Raymond H. P. Crawfurd, M.D.
John Henry Deane.
Percy Furnivall.
Arthur Whitfield, M.D.
Frederick William Forbes Ross, M.D.
Harold Low, M.B.
Charles Ralph Keyser.
George Bertram Hunt, M.B.
Hugh Roger-Smith, M.D.
Alfred Herbert Tubby, M.S.
Charles Lamplough.
H. Campbell Thomson, M.D.
Alfred P. Hillier, M.D., C.M.
F. Sherman Toogood, M.D.
E. Claude Taylor, M.D.
Charles Humphrys.
Charles Sempill de Segundo, M.B.
Arthur John Whiting, M.D.
Alfred Downing Fripp, C.B., M.S.
Chichester Gould May, M.D.
1898 J. J. Perkins, M.D.
James Jackson Clarke, M.B.
Ernest Herbert Cobb.
Sydney Backhouse Hulke.
Thomas Herbert Kellock.
William Darley-Hartley.
Robert Jones.
Arthur Philip Beddard, M.B.
Alfred Ernest Russell, M.D., B.S.
William Cecil Bosanquet, M.D.
Frederick Ingor Willey, M.B.
Jonathan F. C. H. Macready.
Arthur A. H. Partridge, M.B., M.Ch.
Leonard Avery.
Herbert Challice Crouch.
William Turner, M.B., B.S.
Philip Frank, M.D.
Harold Batty Shaw, M.D.
Joseph Stewart Richards, M.D., B.S.

1898 William Henry Breffit Brook, M.D., B.S.
1899 George Edward Rennie, M.D.
W. Warren Low, M.D., B.S.
Rayner Derry Batten, M.D., B.S.
Charles E. Forbes Mouat-Biggs.
Rutherford Morison.
Murray Leslie, M.D.
Herbert Tilley, M.D.
Charles Owen Fowler, M.D.
Cyril Ogle, M.B.
Charles Herbert Fagge, M.S.
Claude Somerville Hawkes.
Henry Stephen Sandifer, M.D., B.S.
Marcus Marwood Bowlan, M.B.
Alfred Eid, M.D.
Karl Fürth, M.D.
Wm. Gream Stone, M.B.
Andrew Duncan, M.D.
Edmond Lucien De Chazal, M.D.
Henry Roe Walker.
Gustave Schorstein, M.B.
Edward Collingwood Andrews, M.D.
1900 Berkeley George Andrew Moynihan, M.S.
Harry Cooper, M.D.
1900 George Augustus Tullis, M.D.
Griffith Lloyd Roberts, M.B., C.M.
Edward Percy Paton, M.S.
Kenneth Scott, M.B.
George Steele Perkins, M.D.
John Arthur Hayward, M.D.
Wilfred J. Hadley, M.D.
John Fawcett, M.D.
Arthur Bousfield, M.B.
J. S. Risien Russell, M.D.
Cecil H. Leaf, M.B.
Josef Paul Zum Busch, M.D.
1901 Ernest George Leopold Goffe, M.D.
Arthur Hamilton Nicholson Lewers, M.D.
Charles Reissmann, M.B.
Adrian Caddy, M.B.
Herbert William Carson.
Thomas Henry Morse.
Laurie Hugh McGavin.
F. U. Menzies.
Thomas Rupert Hampden Bucknall, M.S.
Edward Angas Johnson, M.B.
Percival Horton-Smith, M.D.
Sir Hugh Reeve Beevor, Bart., M.D.

REPORT

OF THE

COUNCIL OF THE CLINICAL SOCIETY,

MAY, 1901.

THE COUNCIL is glad to report that many of the papers read during the Session have been of great value, and the discussions have been well maintained. The alterations in the method of conducting the Clinical Evenings, introduced at the beginning of the Session, have facilitated the exhibition of cases, and, the Council is glad to learn, have met with general approval.

The special feature of the Session has been a discussion on "Enteric Fever occurring among the Troops in South Africa," opened by Dr. Howard Tooth. The Council thought the subject of sufficient importance to justify the publication of the remarks of the various speakers. Two evenings were devoted to this debate, and several officers of the Royal Army Medical Corps—including the Director-General—took part in the discussion.

The question of the disposal of the surplus volumes of the *Transactions* (amounting to no less than 4100 volumes, besides 1358 special reports and 442 indexes) has engaged the attention of the Council. These have been offered to the various Libraries already in receipt of the *Transactions* and requiring volumes to complete their sets; and a similar offer has been made to the Members of the Society, while a sufficient reserve has been retained.

The Council has had under consideration the question of

Composition Fees and the subscriptions of Non-Resident Members, and its recommendations are now submitted to the General Meeting.

The number of Ordinary Members is 594, of Honorary Members 16, giving a total membership of 610. During the Session 20 Ordinary Members have been admitted. The Society has lost through resignation Mr. John Croft, Dr. E. B. Forman, and Mr. Edward Nettleship, while death has removed one Honorary Member, Professor Leopold Ollier, and six Ordinary Members, viz. Sir Henry Acland, Bart., Dr. Julius Althaus, Mr. William Anderson, Mr. Storer Bennett, Dr. Arthur A. Jamison, and Sir William Stokes.

Statement of Cash Receipts and Payments from the 1st May, 1900, to the 30th April, 1901.
HOWARD MARSH, Esq., *Treasurer.*

RECEIPTS.		£	s.	d.
Balance, 1st May, 1900		224	7	4
195 Subscriptions received by Collector	£204 15 0			
198 „ „ Bank	207 18 0			
393		412	13	0
16 Admission Fees at £2 2*s.*		33	12	0
3 Composition Fees (*Transactions*)		15	15	0
Sale of *Transactions*		18	18	0
Sale of Antitoxin Report			1	0
Dividend on Consols (£600)		15	13	8
		£721	0	0

PAYMENTS.				£	s.	d.
Cost of *Transactions*, Vol. XXXIII:						
Printing, paper, binding, delivery, &c.	£163	18	4			
Illustrations	85	11	9			
				249	10	1
Meetings:						
Rent of Rooms	£105	0	0			
A. Tapson (attendance)	7	0	0			
Refreshments	33	8	0			
Hire of Microscopes and Lantern	4	12	0			
Reporting Discussion on Enteric Fever	5	18	0			
Expenses of Demonstration of Instantaneous Radiography	2	13	0			
				158	11	0
Cost of Nominal Transfer of Consols at appointment of New Trustee					19	0
Treasurer and Hon. Secretaries:						
Secretarial assistance	£26	5	0			
Commission to Collector	15	15	0			
				42	0	0
Stationery and Printing	£58	14	2			
Letter of Authority for Sir Dyce Duckworth, Delegate to International Medical Congress	3	13	6			
Preparing Loyal Address to His Majesty King Edward VII	3	13	6			
				66	1	2
Petty Expenditure	9	15	3			
Bank Charges		8	5			
				10	3	8
Balances:						
At Bank	£193	11	1			
Petty Cash		4	0			
				193	15	1
				£721	0	0

R. DOUGLAS POWELL, *President.*
HOWARD MARSH, *Treasurer.*
CHARTERS J. SYMONDS, *Hon. Secretary.*

Examined with the vouchers and found correct.
NORMAN DALTON,
WALTER G. SPENCER, } *Auditors.*

PRESIDENTIAL ADDRESS

DELIVERED AT THE

CLINICAL SOCIETY OF LONDON

ON OCTOBER 12TH, 1900,

BY

SIR RICHARD DOUGLAS POWELL, BART., M.D.,

PRESIDENT.

GENTLEMEN,—I must first of all thank you for the honour you have done me in appointing me for a second year your President, an honour amongst the highest and most prized in our profession.

I did not give any formal address at the commencement of the session last year,—not, I beg you will believe, that I in any way assumed that you would give me an opportunity of doing so this year, but because at that time I felt that I had not the same close knowledge of the later methods of the Society's work as I had of its older lines, and I did not therefore think that any words of mine would at that moment have value in stimulating, or suggesting, or shaping its further course. Here is the thirty-third volume of our *Transactions*, which is certainly equal in interest and value to any that have preceded it; and in these three decades of our work we may find a complete justification of our being, much encouragement for our future, and some hints for our further development.

Sir Thomas Watson, in his inaugural address, 1868, laid it down as the great object of the Society to attain "more exactness of knowledge, and therefore more direct and intelligent purpose and more successful aim, in what is really

the end and object of all our labours—the application of remedies for the cure and relief of disease."

Whilst rational therapeutics must follow in the wake of advancement in pathology and ætiology, it is very remarkable how many of our remedies have preceded in their employment any adequate knowledge of the maladies for which they were used. Thus for nearly four centuries mercury has been used for syphilis, and I doubt if any one could even now tell the precise method of its action. Dr. Withering, 115 years ago, was shown a family receipt for dropsy concocted by an old woman in Shropshire, in which he recognised foxglove as the essential ingredient. Yet, familiar as we have so long been with digitalis, it is only within very recent years that we have had piece by piece explained to us by physiological experiment, the phenomena of slowing and increased regularity of pulse with concentration of force and an increased flow of urine, which bedside observation had taught us to be the beneficial effects of the drug. Cinchona bark has for three and a half centuries been used for intermittent fever, and Fowler's ague drops were employed in the same disease more than a hundred years ago, yet it is only with the discovery of the ague plasmodium by Laveran twenty years ago that the use of quinine and arsenic could be regarded as rationally justified. Again, no one who has any recollection of the rheumatic fever, in the days preceding the introduction of the salicine treatment by Dr. Maclagan in 1874, can fail to appreciate the great value of that therapeutical discovery; yet our knowledge of the exact method of its action is at present but inferential.

We may, then, be thankful for much in empiricism which is but unexplained observation, the well-attested results of which we should be as foolish to reject, because unexplained, as to refuse to profit by the sunshine until we knew the composition of its rays. The great object of this Society is to gain increased knowledge of practical medicine by "the collection of cases, especially such as bear upon undetermined questions in pathology and therapeutics;" and by pursuing it concurrently with the advance of contributory scientific research and discovery we have vastly improved our handling of drugs, often empirically found, in decision and definiteness,—when to employ, how far to push, and when to withhold.

Great strides forward have been made since, twenty-five years ago, Sir W. Jenner spoke here of "the contagious

quality of acute specific diseases" as "a something given off from some part of the sick which can, when properly applied, excite the same disease in another person, and which can excite no other disease." What that something is has been rendered plain to us mostly within the last fifteen years in nearly every one of the acute specific diseases, and in some others which although specific are not acute. Leprosy, pyæmia, dysentery, enteric fever, ague, tuberculosis, pneumonia, diphtheria, erysipelas, cholera, tetanus, syphilis, plague, may be enumerated, the organism specific to each of them having in the sense employed by Jenner been discovered. This exactness of knowledge of the *materies morbi* of these maladies must ultimately give greater precision and effectiveness in treatment, and especially in prophylactic treatment. The report of a committee of this Society on the periods of incubation and contagion of specific fevers is of standard value and authority, and the further inquiry as to the duration of infectiousness in convalescence which must be based upon bacteriological observations is now possible, and might properly be undertaken by this Society. Already some points have been gathered in therapeutics. The triumphs of surgery that have ensued upon increasingly exact recognition of the organisms responsible for suppuration, so fill the mind's eye, that it can scarcely penetrate to the dark ages that ended less than a generation ago. The great terror of the early days of diphtheria has been almost removed by antitoxin treatment—the prophylactic treatment of tuberculosis has been greatly stimulated. A measure of success, encouraging for the future, has at least been attained in tetanus, septicæmic conditions, plague, cholera, anthrax; and we hope this session to receive some valuable contributions presenting for discussion and comparison the experience of those who have, in the African war and in India, watched the results of the preventive inoculations against enteric fever designed by Professor Wright.

Important, however, as are these increased powers of prophylaxis and treatment, the direct fruits of recent combined laboratory and clinical labours, encouraging as they are too for further work, we must yet admit the disappointment that they have failed to help us in some of our more homely diseases, diseases which in so great a degree account for the discomforts and death rates of large communities. The death rate from pneumonia has remained unaltered for fifty years; the influenza bacillus thwarts our best efforts, and carries on

its guerilla warfare year after year with a gay elusiveness worthy of a De Wet; nor can we yet cure a common cold!

In the light of recent combined laboratory and clinical work the ideas of proclivity, hereditary immunity, and epidemic influence have acquired a more definite and practical shape,—nay, have been resuscitated from an oblivion threatened by the natural intolerance of new inquirers for old notions. We are learning how such accidents as shock, chill, pyrexial incidents, local injury, checked secretion, etc., may supply the exciting cause of acute general disease, *e. g.* rheumatic fever, ague, pneumonia, influenza, coryzal catarrh, acute tuberculosis, since in some cases we know and in others we may infer that the organisms specific to such diseases may long dwell with us in abeyance until permitted to assume active cultivation by depression or modification of the vital conditions under which they have been held restrained. The same conditions that lead up to a common cold in an otherwise healthy man would elicit an attack of ague in a fair proportion of persons who harbour the germs of that disease; and, as I have elsewhere hinted, a careful study of the antecedents and bacteriology of a common cold would probably prove rich in illustrations and elucidations of this thesis in its application to many allied diseases. Such an inquiry would, in competent hands, furnish matter for a paper well worthy the attention of this Society, or to form the graduation thesis for a university degree.

The question of artificial immunity must become one of increasing importance to our military, naval, and civil services, as our improving home sanitation renders us increasingly sensitive to diseases which must for long remain endemic in the distant or tropical regions of our vast empire. With our home populations, except for smallpox, the need of artificial immunity is unlikely ever to be felt; and it is to be hoped that the day will even come when, at home at least, we shall no longer need to devitalise our milk and drinking-water, killing the kindly ferments in order to protect us from adventitious and poisonous microbes.

Sir Prescott Hewett, in his Presidential Address in 1872, noted the fact that pyæmia occurred not only amongst the crowded inmates of hospital wards, but also in private patients isolated in luxurious and sanitary houses. Dr. Greenhow—a great sanitary authority in his time—referring in 1879 to Sir Prescott's experience, suggested "that it would seem to indicate some state of the patient predisposing him to the

disease, or possibly some peculiarity of season favouring its development." We smile with the lofty tolerance of more exact information that such inferences should pass muster in this Clinical Society only twenty years ago. Cases, however, I venture to think, come within the experience of physicians and surgeons that would justify Greenhow's shrewd remarks; cases in which incidents of a septic kind, *e.g.* phlebitis and secondary embolism of organs, occur after operations for appendicitis, varicocele, varicose veins, thyroid resections,—not to mention parturition, and operations about the nose and ear passages. In some of these cases which have come under my observation the accidents could be accounted for by no imperfection in the operation, nor in the sanitary surroundings of the patient. The wounds have soundly healed, yet the patient has perhaps an embolic pneumonia or a cardiac embolism. These cases are not very frequent, but one meets with them, and they are more common, if I may judge from my own experience and inquiry, in private than in hospital practice. They are, too, the more striking when met with, since they are relatively more frequent in slight than in very severe operations. My impression is that they are accounted for by an unpreparedness of the patient for surgical injury. The methods of procedure may be aseptic, but the patient in many instances can scarcely be said to be so. In some acute and urgent cases, *e. g.* of strangulated hernia, suppurative appendicitis, and the like, the surgeon has no option but to take the risks and operate immediately. But in chronic cases a few days' preparation, or if necessary a few weeks' regulation of the patient's habits and secretions, would serve to purify him of those effete products of impaired, imperfect, or exuberant tissue change, and inadequate excretion, which cause a morbid reaction to local injury.

In the lifetime of our Society the use of electricity for diagnostic purposes has made great strides; the employment of the electro-magnet for detecting and removing foreign bodies, especially from the eye, of electric light for exploring hollow cavities, and lastly of radiography, have been closely watched and studied here with excellent results for diagnosis; but little, so far, of a direct kind for treatment.

One of the most valuable contributions to the science of medicine to which this Society has largely contributed is the description and thorough elucidation of the disease now known as myxœdema, having been first so named by our esteemed member and late treasurer, Dr. Ord. Sir William

Gull was the first to describe the disease under another name, and it was afterwards thoroughly investigated by Dr. Ord, and reported upon in a special volume of our *Transactions* by a committee presided over by him. Apart from the intrinsic value of investigations which have thoroughly established the nature and treatment of this important disease, the consideration of myxœdema and its treatment by thyroid extract gave an impetus to, if it did not actually originate, the pathological and physiological lines of thought and experimental inquiry which have shown the influence of secretion products absorbed from ductless glands, and from some glands which are not ductless (ovary, testicle), upon mental and physical health, and upon formative symmetry. The sexual glands, the adrenals, bone marrow, spleen, and pituitary body have in this comparatively new light been reinvestigated, and the practical physician is already reaping the fruits therefrom in a greater insight into many previously inexplicable groups of symptoms. The most recent and suggestive outcome in this category is that of the relationship between disease of the pituitary body and that want of growth control which characterises acromegalous disease, of which the first case recognised in this country was brought before this Society by Mr. Godlee.

Another idea that has arisen from the closer association of laboratory and clinical research is the analogy between the affinity of stains for certain tissues and the selective tissue affinity of certain poisons. The affinity, for instance, of the toxins of the tetanus and diphtheria bacillus for nerve tissues, and the very analogous selection of the nervous system by the influenza poison, are rendered tangibly intelligible to us from our laboratory experience of the affinity of different stains for muscle, nerve, fat cell nuclei respectively.

I must not further trench upon your patience by reflections founded upon a glance through the records of work done in this Society, which might be indefinitely extended, and which cannot but bring encouragement and suggestions for present and future work to do.

COMMUNICATIONS.

I.—*A case of Acute Actinomycosis.* By Sir Dyce Duckworth, M.D., and Howard Marsh, *Read October* 12, 1900.

P. D., æt. 19, a lamp-mounter, was admitted to St. Bartholomew's Hospital on September 28, 1899, complaining of much pain, and a swelling on the lower part of the chest.

The patient was a spare, pale, rather undergrown youth, with a sallow skin. Conjunctivæ pearly white. He lay on his back breathing quietly. The tongue was clean, the breath offensive. Temp. 99°, pulse 100, of moderate volume and fair tension. His weight was 6 st. 2 lbs. The chest was badly covered. Respiratory movements equal, except in the left lower region, where a gradual bulging was observable, which became more prominent in the left hypochondriac region and towards the epigastrium, some transmitted pulsation being detectable, visible and palpable.

Lungs.—Right front resonant to sixth rib, where hepatic dulness begins. Breath-sounds natural. *Left front* resonant above, dull in axilla below level of sixth rib. Breath-sounds natural over the resonant area, but absent over the dull area. Posteriorly: *Right back* resonant, with good respiratory murmur. *Left back* resonant above, but dull for four inches at the base. Respiratory murmur good above, impaired below, with some friction crepitus.

Heart.—Apex-beat visible in fourth left space, half an inch within the nipple line. Cardiac dulness extended from the third left costal cartilage as far as the level of the fifth down the left border of the sternum, where it joined that of

the left lobe of the liver. The sounds were loud and clear. The abdomen moved with respiration, and was not generally distended. The bulging described above constituted partly a thoracic and partly an abdominal tumour, and appeared to involve the left lobe of the liver very materially. The tumour was dull on percussion and very tender on palpation. It descended to some extent with the action of the diaphragm, suggesting hepatic involvement. The edge of the right lobe of the liver was not to be felt below the costal cartilage. The spleen was palpable, apparently continuous with the hepatic mass. No other viscus or mass to be felt.

The *urine* was of sp. gr. 1026, and void of albumen. The knee-jerks were exaggerated. There was no history of any injury having been received. There was a history of pain in the stomach which had occurred after taking food some months previously, and this appeared to indicate that there had possibly been a gastric ulcer at that time. This symptom had passed off. The apparent duration of the illness may be reckoned as not exceeding six months. The temperature rose to 99·6° on the day of admission, and was normal and subnormal on the 24th September. On the 25th it rose to 102·2°, and on the 26th to 102·8°.

The patient was clearly very ill, and suffering much. His symptoms and physical signs betokened the presence of a large abscess either in or in connection with the left lobe of the liver. In the absence of the usual antecedents or concomitants of this condition, it appeared strange that such should have occurred.

The temperature having risen on the 25th, the pulse being 140, and the patient flushed and sweating, it was decided in consultation with Mr. Bowlby, acting in Mr. Marsh's absence, to puncture the swelling and search for pus. A long exploring needle was therefore passed through the epigastrium into the left lobe of the liver. No pus being obtained, a second puncture was made through the eighth right intercostal space in the anterior axillary line, but without the exit of any fluid. The following day the upper border of the cardiac dulness was found to be a finger's breadth higher, beginning at the upper border of the third costal cartilage. Having experience of some similar cases, I ventured to suggest the possibility of this condition being one of actinomycosis involving the liver and adjacent parts.

As the patient now required more surgical than medical treatment, I sought the aid of my colleague, Mr. Marsh, who

suggested further exploration with the object of alighting upon some purulent focus. This was very thoroughly carried out under an anæsthetic in several situations, but without any result. Small fragments attached to the needle were examined with a view to discover any evidence of ray fungus, but no trace of this was obtained. I therefore transferred the patient to Mr. Marsh's care, and he was removed to a surgical ward on September 28.

No further operative interference was adopted for the next few days, but on October 6 laparotomy was performed, and a swelling situated below and behind the left lobe of the liver was fully explored, but no pus was reached. Subsequently the patient quickly lost flesh and strength; the temperature rose to 102°—103°, and several severe rigors occurred. As, however, the local condition underwent no material change, no more exploratory proceedings were undertaken. He died on November 7.

This case was obscure from the beginning, and, indeed, throughout its course. The early symptoms indicated the probability of gastric ulcer, a condition, however, not common in a young male adult. There was a prominent tumour present on the patient's admission to the hospital, and the position and features of this appeared significant either of hepatic abscess involving the left lobe of the liver, or of suppuration in connection either with that organ or the left pleural cavity. The absence of any history of dysentery or of tropical influences rendered the idea of any form of hepatic abscess unlikely. The symptoms connected with the gastric ulcer had passed off for some time before the patient applied to the hospital, and hence attention was diverted from that chapter in the history of the case. The autopsy revealed the true nature of the disease, and confirmed the early surmise as to the existence of actinomycotic invasion.

A softened patch was found near the cardiac end of the stomach, and a probe readily passed through it into a mass of spongy actinomycotic growth in the left lobe of the liver. It remains a question as to the possibility of the fungous elements having originally entered the textures by this channel, for this is a course which is now well recognised in such cases, a breach of mucous surface affording a ready entry for such intrusion. The growth spread indiscriminately, and so came to invade the liver, diaphragm, pericardium, both lungs, and other parts. There was a subphrenic abscess of considerable size, and the pus contained in it was of a yellowish-

green hue, having dispersed in it numerous dark green, almost black granules, looking much like coarse gunpowder. These proved to be masses of ray fungus. Cultivation of the pus showed no microbes of septic or other characters. This is noteworthy, while remembering that there was prolonged pyrexia with occasional rigors. It was unfortunate that no clear evidence was obtained during life of the true nature of the process, for it would have been proper to employ large doses of iodide of potassium with a view to destroy the invading organism. There is a difficulty in believing that the gastric ulcer, if it really existed, was the original channel of infection, inasmuch as it is not likely that other infective matters could have failed to enter together with the specific fungous elements, and none of these were found.

The exact course followed in this instance must, therefore, remain undetermined. I reported a case in some respects of similar nature in the *St. Bartholomew's Hospital Reports* for 1895,* one of actinomycosis of the thoracic wall, in which no clue to the mode of infection was obtained. Both cases well illustrate the fact that in man it is common to meet with a form of burrowing suppuration as the result of this invasion, while in the lower animals more localised tumour formations are found.

* Vol. xxxi, p. 23.

II.—*The sequel to a case of Hydatid of the Liver: showing also the results of abdominal section in Ascites.* By NORMAN DALTON, M.D. *Read November* 9, 1900.

THE patient, L. N., was a woman of 23 at the time of her death in 1899, but her illness began in 1883, when she was aged 7. I first saw her in the out-patient room at King's College Hospital with Dr. Hayes in 1892. She was then suffering from ascites, and as she was thin and tubercular-looking, our first idea was that the case was one of tubercular peritonitis. However, either then or on some subsequent examination, we thought that we could feel the liver and the spleen through the ascitic fluid, which suggested that the real condition was cirrhosis of the liver beginning in early life. In the meantime I had written to Dr. Barlow, under whose care the patient had been in Great Ormond Street Hospital when she was a child; and he replied to the effect that she had at that time suffered from a hydatid cyst of the liver. She had first of all been aspirated, with the result that hydatid fluid was found. Then, after a considerable interval (apparently in 1887), she returned to Great Ormond Street with jaundice and swelling of the liver. On this occasion, while in the hospital she passed hydatid cysts *per anum* (with relief to the jaundice), but developed pneumonia at the right base, with severe hæmoptysis and the subsequent abundant expectoration of bile. The case was watched for a long time, and the spleen was observed to get larger as the liver got smaller, but there was at that time no ascites. Apparently the patient was quite well from about 1888 until the ascites developed in 1892.

The ascites soon required tapping, after which operation the liver and spleen could be distinctly felt. The fluid rapidly re-accumulated, so that paracentesis had to be performed frequently, and towards the end of the summer of 1893 she required tapping every fortnight. As this state of affairs must soon have ended fatally, I advised an exploratory abdominal section, in the hope that a new hydatid or some remains of the old cyst might be found and removed or emptied. It was rather a forlorn hope, because the absence of

jaundice indicated that there was nothing of much size in the portal fissure, and that the portal obstruction was therefore intra-hepatic. Still, the other alternative was certain death. Mr. Watson Cheyne concurred in my views and performed the operation. Unfortunately nothing was found which could be removed. There were some adhesions on the convex surface of the liver round the old perforation into the lung, but otherwise there were no adhesions on the liver, or on the stomach, or on any part of the upper portion of the peritoneal cavity. This is important in the light of subsequent events. The gall-bladder was not seen.

The operation was performed at the end of October, 1893, and as no change in the condition of affairs had been effected by it, I naturally expected that the ascites would return, and considered that the case was now hopeless. The wound healed well, and on November 5th paracentesis became necessary, ten pints being withdrawn. On November 13th the paracentesis had to be repeated, nine pints being withdrawn; but after this the fluid did not return with any rapidity, and she went home to convalesce. This was at the end of 1893, and I did not examine her again until some time in 1898. But I saw her mother several times, and she told me that the fluid had not returned, that the girl did not look unnaturally large, and appeared quite well, being able to bicycle long distances. At the end of 1898 she had some illness like influenza at home, and then expectorated bile, showing that the old fistula between the liver and the lung was still patent. When she was better she came to the hospital, where I saw her. I found that although the ascites was practically cured, there remained a localised collection of fluid between the umbilicus and the pubes, quite central, and not altering its position with that of the patient. It therefore seemed clear that the cure of the ascites was due to adhesions which had obliterated the peritoneal cavity, except in the lower umbilical and hypogastric regions. It is certainly remarkable that the patient should have been so well during those five years, and in particular, that no hæmatemesis or melæna should have occurred. In the erect position there was a good deal of ventral hernia at the operation wound. I warned her against exertion, but she did not take my advice. The end came in May, 1899. She had attempted to lift a heavy box, and was seized with great pain in the abdomen and collapse. From this she rallied, and was brought to the hospital. For a day or two she exhibited the symptoms of

peritonitis, but appeared to be improving. There was no intestinal obstruction. However, on the fourth day the temperature became higher and her condition generally worse. Crepitations could be heard at the right base, and she expectorated a good deal of bile. An exploratory operation was attempted, but as I had surmised, the peritoneal cavity was found to be obliterated by old adhesions except in the hypogastrium. Death occurred on the eighth day.

At the *post-mortem,* old fibrous adhesions were found between all the viscera in the upper two thirds and the sides of the abdominal cavity. In particular, the intestines were adherent to the operation wound.

There were, however, numerous small loculi between the adhesions, and these were filled with pus. In addition to the adhesions there were other signs of chronic peritonitis, for the mesentery was short and thick, and the omentum was rolled up to form a cord. The cavity in the hypogastrium, due to the absence of adhesions there, was filled by turbid serum, and its walls were much inflamed. The small intestines, especially those which formed the walls of the cavity, looked as if they had all been strangulated in a hernia, *i. e.* they were dark red in colour, covered by swollen veins and subperitoneal hæmorrhages, with pus and fibrin on the surface. No strangulation, however, could be found, and the lumen of the bowel was everywhere patent. I therefore came to the conclusion that when lifting the heavy weight a large mass of intestines must have occupied the ventral hernia at the abdominal wound, and been there so compressed, that thrombosis of a large number of veins occurred. The compression of the bowel would soon be relieved, but the thrombosis of the veins eventually caused a septic peritonitis. There was no perforation. The spleen was very large and soft; it weighed twenty-three ounces, and was pushed up so as to encroach greatly on the thoracic cavity.

As regards the hydatid, we found a small cavity containing calcareous particles in the upper part of the liver, just to the right of the falciform ligament. Below, it communicated with a dilated bile-duct, and above with a fistula (shut in by adhesions) which went through the diaphragm, and entered a dilated bronchial tube in the lower lobe of the right lung.

Glisson's capsule was very greatly thickened, and the peritoneum over the gall-bladder formed a continuous membrane with it, so that the gall-bladder was completely

flattened against the liver, and quite invisible until its coverings were cut through, which explains the fact that we did not see it at the operation. Microscopically it appeared that there was a chronic interstitial hepatitis, due to an ingrowth of fibrous tissue from Glisson's capsule into the connective tissue between the liver lobules; because, whereas sections of the liver near to Glisson's capsule show extreme cirrhosis, those which were taken from the middle of the liver show only slight cirrhosis.

It is very remarkable that this patient should have escaped, at the age of 11, from the peril of a hydatid bursting into the lung; should have, again, at the age of 17, survived the danger of an ascites, the cause of which was traceable to the hydatid, and should finally have succumbed, at the age of 23, to an accident which was due to the state of affairs left by the original disease. It is curious to note that, although the fistula from the liver to the bronchus had never been closed by sound fibrous adhesions, bile appears to have been expectorated on only two occasions, viz. when she had what was called influenza, and when she was dying of peritonitis, on both of which occasions fever was present. While she was in a state of exhaustion from ascites there was no expectoration of bile. But the chief interest attaches to the extensive chronic peritonitis and formation of adhesions which followed the operation. We know that they did follow the operation, because they were not present when the abdomen was opened, and because the ascites ceased to reaccumulate a fortnight after the operation; but it is difficult to say why they occurred. There was no acute peritonitis, for the wound healed well without pain or fever. Neither can the adhesions be accounted for by the irritation produced by handling, for only the parts of the peritoneum near the liver were touched, while the thickening and adhesions were wide-spread. It is remarkable, however, how far the growth of fibrous tissue may spread by contiguity beyond what we would imagine to be the centre of the irritation. For instance, it has often struck me as strange that in cirrhosis of the liver not only may Glisson's capsule be thickened, but the falciform ligament may be extremely thick and contracted. Now, supposing that the original cause was the irritation of the connective tissue of the liver by alcohol, it is difficult to see why the falciform ligament outside should be irritated, unless we suppose that fibrous thickening of the capsule became, in its turn, a source of irritation to the ligament.

Still, we know from experience that peritoneal adhesions do not usually become generalised, and I am led to believe that the two factors which produced the extensive adhesions in this case were the admission of air at the operation, and the condition of the peritoneal surface caused by the ascites. We know that the admission of air by itself would not have been sufficient, because after aseptic abdominal section few adhesions or none at all are found. But when we consider that in ascites the endothelium of the peritoneum is sodden and ill-nourished, and is often shed in great quantity, it seems possible that the exposure to the air may have determined sufficient detachment of endothelium to have caused extensive adhesions to form, and that the whole process may have taken place so quietly as to have caused no symptoms of acute peritonitis. In favour of this view is the fact that cases of tubercular peritonitis in which ascites is present are often cured by abdominal section, and in such cases it is probable that extensive adhesions take place. At any rate, I think that this case should encourage us in treating the ascites of cirrhosis of the liver by abdominal section, in fact, the case is practically one of cirrhosis, because the hydatid had become quiescent.

The last point for consideration is the manner in which the adhesions stopped the ascites. One's first idea would be that, by obliterating the peritoneum, they mechanically prevented the accumulation of fluid therein. But if that were the case, one would have expected that the portal congestion would have been relieved by hæmatemesis or melæna. So that I think that the adhesions really relieved the portal congestion itself; and it is easy to see how they could do so, for the vessels of the adhesions would directly unite the mesenteric and other portal capillaries with the capillaries of the veins in the anterior abdominal wall, diaphragm, etc., by which veins the blood could return freely to the heart.

III.—*On the Diastolic Cardiac Sound which causes Spurious Reduplication of the Second Sound at the Apex, and is sometimes called the Third Sound of the Heart.* By T. Stacey Wilson, M.D. *Read November* 9, 1900.

I WISH to bring before you this evening several cases where this sound was well heard, because I think they throw important light upon the mode of occurrence and the clinical value of this diastolic cardiac sound.

But before describing the cases I must discuss briefly what I believe to be the cause and the meaning of this sound. I take what I consider to be the generally accepted view, that this spurious reduplication of the second sound is due to the appearance of an additional sound after the true second sound, and that this sound is in some way produced by the entry of blood into the ventricle at the commencement of diastole. This view was ably advocated twenty years ago by Dr. Sansom, and is well supported by arguments in his book on the *Diagnosis of Diseases of the Heart.* I need not speak of the clinical features of this sound; they have been often described, but nowhere in my opinion more clearly and accurately than by Dr. Arthur Phear in the *Lancet* for 1897, p. 97. With him I agree that so definite and distinct a sound ought to have a name of its own. It has been called the third sound of the heart, and I do not think a more appropriate name could be found for it, and for the sake of clearness and brevity I must ask to be allowed to call it the third sound of the heart in this paper.

The sound to which I refer in this paper is a short, sharp sound. I do not refer to the mitral diastolic murmur.

The theoretical part of this paper involves so many points upon which proof may rightly be asked for, that I have found considerable difficulty in condensing all I have to say into the space at my disposal. The ground is, in part, covered by a paper upon the diastolic expansion movement of the ven-

tricles which I read at the Ipswich meeting of the British Medical Association (*vide Brit. Med. Journ.*, September 29, 1900). For the sake of brevity I have put the theoretical part of my paper into the following series of propositions.

1. The diastolic expansion of the ventricles plays so important a part in the circulation that it must be reckoned as a distinct phase of the cardiac cycle (*vide Brit. Med. Journ., loc. cit.*).

2. Record of the second cardiac sound on the cardiogram shows that the second sound occurs at or just after the commencement of this expansion movement (*vide* Cardiograms, Case 2, figs. 1 and 2; Case 5, figs. 1 and 2; and Case 6).

3. Record of the so-called third sound on the cardiogram, and careful measurement of the heart-sounds and tracings, show that the third sound occurs at or just before the commencement of the period of complete muscular relaxation (*vide* Cardiograms, Cases 3, 5, and 6).

4. Therefore the third sound of the heart occurs at the end of the expansion movement, and marks the commencement of the third phase of the ventricular cycle. It therefore resembles the first and second sounds in marking the commencement of a distinct phase.

The first sound occurs at the commencement of the ventricular contraction.

The second sound occurs at the commencement of the ventricular expansion.

The third sound occurs at the commencement of the ventricular relaxation.

5. The duration of the expansion movement is usually about one tenth of a second, *i. e.* from about half to two thirds as long as the ventricular systole (see tables of measurements on page 12).

6. In cases where the third sound is well marked there is often to be seen in the cardiogram a short sharp rise, immediately following the fall due to the muscular relaxation. This oscillation I propose to call (from my belief as to its origin) the suction recoil wave.

7. This wave varies somewhat in character. It may—

(*a*) Be only or mainly visible in tracings taken at the apex, and may be more or less due to a tilt forward of the relaxed anterior wall of the heart, such as might be due to the impact against it of a current of blood entering the ventricle from the auricle.

(*b*) In other cases it is disproportionately greater the

Measurements of Phases of the Cardiac Cycle.

Measured in $\frac{1}{20}$ths of a second.

	Measurements of Sounds.*						Measurements of Cardiogram.				
						Average.					Average.
Case 2.											
Systole	3	3½	3	2½	—	—	4	4½	4	4½	4·2
Expansion . . .	—	—	—	—	2	—	2	2	2	1¾	1·96
Total beat . . .	10	11½	10½	9½	9	—	11	11	10	10	10·48
Number of beats measured . .	—	—	—	—	—		7	7	10	5	
Case 3.											
Systole	3½	3½	—	—	—	3½	3¾	4	4½		4·15
Expansion . . .	—	—	2½	2	1⅘	2	2	2	2		2
Total beat . . .	12	12	12	11	11	11·6	12	11	11½		11·4
Number of beats measured . .	7	13	6	7	7		6	11	12		
Case 5.											
Systole	5	5	—			5	5½	6			5·8
Expansion . . .	—	—	2	1½		1·8	1½	2			1·8
Total beat . . .	14	12	14	2		13·2	12	14			13·2
Number of beats measured . .	8	5	5	4			6	9			

more rapid the previous fall of the lever, and might be largely mechanical or of the nature of a rebound of the lever. (As this rebound does not occur to the same extent in the normal heart, or in simple mitral regurgitation, it seems to have some pathological significance, and to imply an abnormal

Explanation of the Plates.

The tracings here shown were all taken with a Galabin's cardiograph, and are to be read from left to right.

In all the tracings except Case 4, Figs. 8—11, and Case 5, Fig. 13, the position of the cardiac sounds as marked on the cardiogram was ascertained by synchronising the raps of an electric signal with the heart sounds, and in this way obtaining a simultaneous record of the heart sounds and the cardiogram. The actual record of the heart sounds is shown in relation to the cardiogram in Case 2, Fig. 3; Case 4, Fig. 7; and in Case 5, Fig. 12. In all other cases the actual record is not shown for want of space.

Enlarged photographs of the tracings in question were, however, shown to the members of the Society when this paper was read before them, and the accuracy of the interpretations here given was demonstrated.

In Case 4, Figs. 8—11, the theoretical position of the first, second, and third sound has been marked.

* The time between the first and second sounds is reckoned as the systole although the first sound occurs just after its actual commencement. The time between the second and third sounds gives the duration of the expansion movement.

In Case 5, Fig. 13, the theoretic position of the second sound as marked in the figure was verified by other combined tracings from the same case.

CASE 1.—MITRAL STENOSIS WITH A WELL-MARKED THIRD SOUND.

FIG. 1.—Tracing taken at the apex, which was in the fifth interspace, 2½ inches from the sternum. Record of the first and second sounds on the cardiogram. In the case of the third beat the sound record is faulty owing to the irregularity of the beat. The third sound would occur at or just before the lowest point of the fall after the second sound. The suction recoil waves following the fall are well shown, and their relationship to the long pause demonstrates them to be of ventricular and not auricular origin.

FIG. 2.—Tracing similar to Fig. 1, but taken from near the apex and when the heart was acting excitedly. The suction recoil waves are higher than in Fig. 1. The sharp wave just before the main systolic rise is probably of auricular origin.

excess of intra-ventricular tension at the moment when it occurs.)

(*c*) In other cases this suction recoil wave is observable in tracings taken from every available part of the ventricle wall, and is evidently due to a sudden increase in the volume of the ventricle, such as could only occur by the sudden entry of blood with considerable force (*vide* Cases 2, 3, and 4).

8. In some cases therefore where this third sound occurs, we seem to have evidence of some increase of tension within the ventricle just after the onset of ventricular relaxation, and sometimes we see an actual general expansion of the ventricle at this point of time.

9. The cardiograms show that this increase of tension is due to the preceding activity of the ventricles, for the suction recoil wave is separated from the following auricular systole by an interval which varies with the varying length of the long pause (*vide* Cases 1 and 3).

10. We may therefore conclude that this distension of the ventricle is due to the entry into it of blood which was set in motion by the ventricular aspiration developed during the preceding expansion movement. (The other alternatives would be to believe in a second ventricular systole in the middle of the long pause, or to believe in an early and wholly unprecedented form of auricular activity. There is no evidence at all in favour of either of these.)

11. We therefore can say that where the third sound is well marked there is evidence that blood is still flowing into the ventricle in a more or less powerful stream after the complete relaxation of the muscles has set in.

12. It will, I think, be readily admitted that in the case of the normal heart the size of the mitral orifice is so considerable, in comparison with the cubic capacity of the auricle, that a powerful ventricular aspiration, lasting half as long as the systole, would suffice to draw all the contents of the auricle into the ventricle by the time the aspiration ceased.

Therefore we may say that in the normal heart all movement of blood due to ventricular aspiration will practically have ceased by the time relaxation occurs.

13. As a result of the above considerations I have advanced the following suggestions as to the mode of production of this sound.

I. The third sound of the heart is not produced by the first inrush of blood into the ventricle. (In some cases of

mitral stenosis I believe that a sound or a murmur may be produced at the mitral orifice by this first inrush, but it is, of course, synchronous with the second sound.)

II. During the expansion movement the segments of the mitral valve are, presumably, kept tense by the activity of the expanding muscles, but on the onset of relaxation they are free to move. If, therefore, a powerful enough stream of blood is still flowing through the mitral orifice at the onset of relaxation, it will be apt to move the valve segments with an audible sound at this point of time.

III. If my view is correct, the occurrence of a third sound is evidence that the auricle is unable to empty its contents into the ventricle as rapidly and completely as normal.

IV. This prolongation of the flow will occur—

(1) Where there is a narrowing of the mitral orifice; or

(2) Where the auricle holds more blood than the ventricle can draw through the normal mitral orifice during the expansion movement.

V. We may, therefore, have a third sound, occurring—

(1) In mitral stenosis.

(2) Where the auricle is so dilated as to hold more blood than the ventricle can.

(3) Also, probably, where the muscle of the ventricle is too weak to develop an amount of aspiration sufficient to empty the auricle as rapidly as normal.

The cases I wish to refer to belong mostly to the second of the three last-named groups, but I shall first show two cardiograms from the apex of a case of ordinary mitral stenosis with a well-marked third sound. They both show (as is usual in mitral stenosis) an absence of any expansion at the time of occurrence of the second sound (compare Case 5, fig. 12). After the fall of the lever which marks the onset of relaxation there is a sudden sharp rise, followed by a second or a third. These are well marked in Fig. 2, which was taken on the outer side of the apex, but when the heart was acting excitedly. On this tracing I did not record the heart-sounds. I have not marked the position of the third sound on this cardiogram, but it would occur at the foot of the fall just before the recoil waves.

Case 2.—This is the case to which I wish to draw special attention this evening.

It was the case of a girl æt. 12, admitted to the Birmingham General Hospital suffering from pericarditis and rheu-

matic pains, with a history of acute rheumatism eleven weeks previously.

On admission she was a pale, poorly-nourished child, with considerable dyspnœa but no dropsy. There was well-marked pericardial friction all over the cardiac area, and a loud mitral systolic murmur, which was well conducted to the axilla and back. It was specially loud posteriorly in the left mid interscapular area, and was well conducted down the spinal column, being audible as far as the sacrum. The apex was, however, not displaced outwards or downwards, being in the fifth interspace in the nipple line. There was a loud third sound. The aortic valves were healthy. There was a slight amount of tricuspid regurgitation with some pulsation of the veins of the neck, also some slight enlargement of the liver, but no pulsation. There was no marked dilatation of the heart to the right, and none of the ordinary signs of failure of the right side of the heart.

She remained in much the same state for a month or more, confined to bed with considerable dyspnœa and marked anæmia. The increase of the anæmia was accompanied by marked upward dilatation of the right ventricle, and the shock of the pulmonary valve closure could be felt high up in the second interspace some one inch from the sternum.

After about five or six weeks she began to show signs of ulcerative endocarditis, and she also began to have attacks of anginous pain.

Throughout the case the symptoms were more those of aortic disease with failing forward pressure than of the ordinary mitral type, and it was clear to me that something was preventing the ordinary process of compensation from taking place. The anginous attacks were very similar to the ordinary ones of aortic disease, but of no very great severity, and not accompanied by any great excess of dyspnœa, nor by the usual sense of dread. She gradually got worse, and died rather suddenly of simple cardiac failure. There was at no time any dropsy or engorgement of veins, nor much cyanosis.

Cardiograms taken from any part of the heart showed an extremely well-marked suction recoil wave. In those taken at or near the apex there was a sharp-pointed wave, just following the cessation of ventricular activity, which rose as high as the systolic eminence itself (*vide* cardiograms, Case 2). The third sound would come in the hollow just before this recoil wave.

The diagnosis was that there was very considerable mitral

regurgitation, with a dilated left auricle, because of the loudness of the heart-sounds in the interscapular area and down the spine; also in view of the loudness of the third sound (and the presence of an occasional diastolic murmur) mitral stenosis was suspected. The pericardium was probably adherent.

At the *post-mortem* there was, however, no narrowing of the mitral orifice. It was of normal size, but its cusps and cordæ were thickened, and it was evidently incompetent.

CASE 2.—MITRAL REGURGITATION WITH A WELL-MARKED THIRD SOUND (AS WELL AS THE SYSTOLIC MURMUR).

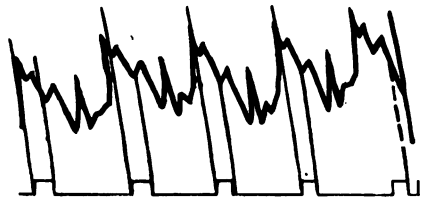


FIG. 3.—Record of the first and second sounds on a cardiogram from near the apex (taken in the fifth interspace, 2 inches from the sternum). Note the prominent and pointed suction recoil wave occurring some time after the second sound.

FIG. 4.—Tracing taken from the third left interspace, 1¼ inches from the sternum. The timing of the sounds is not quite accurate. The positions of the first and second are recorded. Note the presence of a suction recoil wave in this tracing taken over the conus arteriosus of the right ventricle.

There were recent small vegetations on the aortic and mitral valves. In spite of the regurgitation the ventricle was of normal size, its cavity holding 90 c.c.; the left auricle was, on the other hand, very greatly dilated, holding 164 c.c. The other chambers were not specially dilated.

The explanation of the case is, I believe, as follows:—The pericarditis preceded the mitral disease, and the support given by the adhesion of the pericardium prevented the left

ventricle from dilating sufficiently for compensation to become established. It also prevented the right side from failing, and therefore great strain was thrown upon the left auricle, causing its dilatation. The pallor, dyspnœa, and absence of dropsy were due to the small amount of blood which the heart could put in circulation, losing, as it did, a considerable portion of a normal charge at each beat, owing to the regurgitation.

This case gives us a clear demonstration of the process of compensation when we read the clinical signs and the cardiograms in the light of the post-mortem.

We see the reason for the extreme suction recoil wave. We have a ventricle holding only 90 c.c., aspirating with all its force at an auricle holding 164 c.c. At the end of the expansion movement of the ventricle, when the ventricle has, in virtue of its elastic and its muscular recoil, drawn all the blood into itself which it can hold, there is still nearly as much again in the auricle waiting to come in. This has already been set in motion by the ventricular aspiration, and its momentum carries it on towards the ventricle, causing its dilatation, and making the sudden rise in the cardiogram. If the walls of the ventricle had not been unduly resistent (owing to the pericardial adhesion) this distensile force would long ago have dilated the ventricle till it was large enough to completely empty the auricle during its expansion movement, and so re-establish normal conditions. It could not do so, however. The strong rush of blood into the ventricle after the onset of relaxation would be amply sufficient to cause the loud third sound heard during life, and the prominent recoil wave in the cardiogram.

This case has given me a clear demonstration of the means whereby compensatory dilatation of the ventricle is established in mitral regurgitation, and I feel much gratitude to that little pointed recoil wave in the cardiogram.

Case 3.—The next set of tracings are from a child who had pericarditis, and whose symptoms were very similar to Case 2, but whose heart was greatly dilated. There was a systolic mitral murmur, a well-marked third sound and diastolic murmur.

The suction recoil wave was most prominent here. She probably had a dilated left auricle, as in Case 2. She died of sudden heart failure, but unfortunately no post-mortem examination could be obtained.

In tracings Nos. 5 and 6 the first and second sounds are recorded on the cardiogram, and in No. 7 the second and third sounds only.

CASE 3.—MITRAL REGURGITATION WITH A WELL-MARKED THIRD SOUND.

The apex was in the seventh interspace in the axilla, about 4½ inches from the sternum.

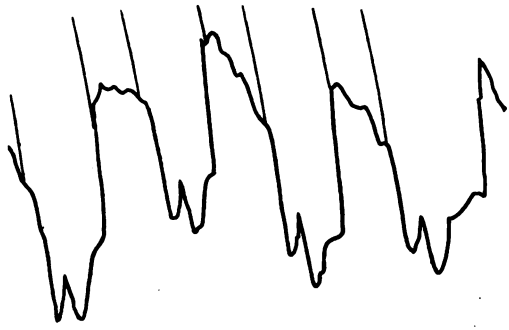


FIG. 5.—From the sixth interspace, 4½ inches from the sternum. A record of first and second sounds on the cardiogram is shown. The suction recoil wave is seen after the diastolic fall of the lever of the cardiograph.

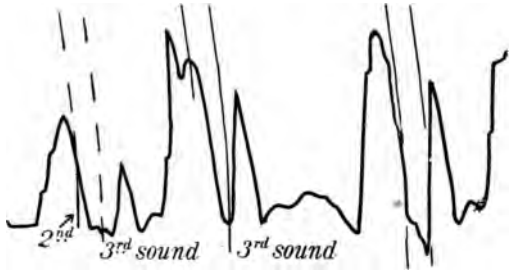


FIG. 6.—From the seventh interspace 4 inches from the sternum (*i. e.* nearly one inch internal to the apex-beat). Record of second and third sounds on the cardiogram is given. Note the very high suction recoil wave which follows the third sound. The timing of the third beat is not quite accurate owing to the irregularity of its interval after the preceding beat.

CASE 4.—The next set of tracings are from a woman, æt. 46, who suffered from palpitation, swelling of the legs, and occasional dyspnœa. The interesting feature about the case was that the third sound was much louder than the first

sound. The heart was extremely difficult to time, because the attention was at once arrested by the second and third sounds, and they appeared to be first and second. There was a faint presystolic murmur and a faint first sound. On taking cardiograms it was found that there was an extremely prominent recoil wave. In some parts of the heart it was the most prominent part of the cardiogram. It was present

CASE 4.—PATIENT WITH A VERY LOUD THIRD SOUND, A FAINT FIRST SOUND, AND A VERY FAINT PRESYSTOLIC MURMUR.

A very prominent suction recoil wave is seen in tracings taken from all parts of the left and right ventricles.

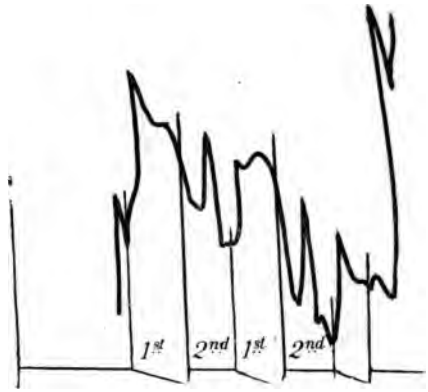


FIG. 7.—Record of the first and second sounds on the cardiogram. Note the prominent suction recoil wave occurring some time after the second sound. The irregularities of the tracing are due to respiratory movements of the chest.

in all tracings. I show tracings from the apex, the fourth interspace one and a half inches out, the fifth space three and a half inches out, and from the epigastrium. The explanation of this recoil wave appearing in tracings taken over the right ventricle is probably to be found in the movement of the interventricular septum.

CASE 5.—I show two tracings from a woman with compensated mitral regurgitation with some stenosis. The tracings show the prominent expansion movement characteristic of mitral regurgitation. There was also in this case a well-marked third sound, and a diastolic murmur following it.

In this case the third sound occurs just before the foot of

the fall of the apex tracing, and its place is often marked by a small oscillation in the downstroke. I would suggest, as an explanation of this, the possibility that the musculi papillares relax slightly before the rest of the ventricle wall, and so admit of the third sound occurring just before the onset of general relaxation.

Fig. 8 is a cardiogram taken just internal to the apex

FIG. 8.

FIG. 9.

FIG. 10.

FIG. 11.

FIG. 8.—From the apex (in the fifth interspace, 4 inches from the sternum).
FIG. 9.—From the fourth interspace, 1 inch from sternum.
FIG. 10.—From the third interspace, 2½ inches from sternum.
FIG. 11.—From the epigastrium.

In these tracings the approximate theoretic position of the heart sounds are shown. The position of the third sound is marked by a broken line.

with a record of the second sound marked on it. It shows the systolic depression in the cardiogram, followed by a well-marked expansion wave. The second sound occurs near the top of this wave.

CASE 5.—MITRAL REGURGITATION AND STENOSIS WITH A WELL-MARKED THIRD SOUND.

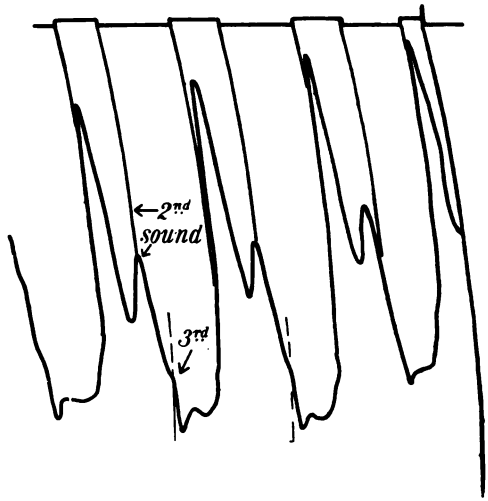


FIG. 12.—Tracing from the apex, showing a record of the first and second sounds on the cardiogram, and the theoretic position of the third sound as verified by other tracings.

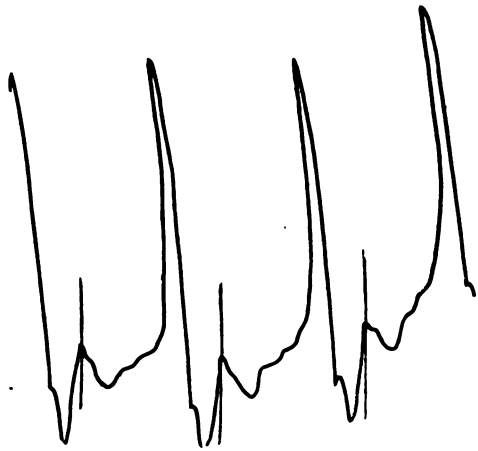


FIG. 13.—Tracing taken internal to the apex where the diminution in the volume of the ventricle at the end of the systole causes a fall in the tracing. The position of the second sound is shown and the rise due to the diastolic expansion movement is seen. The third sound would occur in the following notch.

CASE 6 shows a cardiogram from a case with a well-marked third sound, and is a record of the second and third sounds on the cardiogram. It shows the third sound coinciding with a small oscillation near the foot of the diastolic fall of the tracing.

CASE 6.—ANÆMIC DILATATION OF THE HEART WITH A WELL-MARKED THIRD SOUND.

FIG. 14.—Tracing from the apex showing a record of the second and third sounds. A slight broad suction recoil wave is seen following the third sound.

Before concluding this paper I must express my indebtedness to my house physician, Dr. Cyril Lewis, whose patience and skill in manipulating the cardiograph while my attention was taken up in recording the heart-sounds, has rendered it possible for me to obtain the satisfactory combined tracings which I figure in this paper.

In conclusion, I am conscious of the difficulty in a short paper of adequately and briefly justifying the theoretical considerations which I submit, but of which I have what I consider abundant proof. Nevertheless, the facts of Case 2 speak for themselves, and tell us much as to the conditions which determine the appearance of this third sound of the heart.

In spite of its shortcomings, I hope that the clinical research which I have laid before you this evening may tend to an increase of our knowledge upon this most important but difficult subject of the expansion movement of the ventricles.

IV.—*A case of Thrombosis of the Inferior Vena Cava and of the Iliac and Femoral Veins, of acute onset and fatal termination, with symptoms resembling those of Perforated Gastric Ulcer.* By SIDNEY PHILLIPS, M.D. *Read November* 23, 1900.

B. P., a maid servant, æt. 19, after having indigestion for a long time, began about the middle of November, 1899, to suffer pain in the epigastrium after food; though compelled by the pain to be careful in her diet she did not seek medical advice. There was never any vomiting, hæmatemesis, or melæna.

At 10 o'clock on the morning of the 29th November, 1899, while at work, she was suddenly seized with pain in the epigastric region, so severe that she became collapsed, took to her bed at once, and was seen by a medical man, who sent her into St. Mary's Hospital under my care on the next afternoon. She took a small quantity of milk by the mouth between the time of onset of the pain and her admission into hospital. She had vomited once.

I saw her on the evening of admission, November 30, at 9.30 P.M.; she was a very anæmic girl, with great beads of sweat upon the face; the temp. 103°; pulse 120 per minute. The abdomen was moderately distended, but its respiratory movements were much restricted; she complained of pain in the upper part of the abdomen; there was local tenderness about 1½ inches below the tip of the xiphoid cartilage, and a little to the left of the linea alba; the rectus muscle was kept tense in this situation.

The area of liver dulness was not lessened, and there was stomach resonance as high as the fourth rib in the left mid-axillary line and downwards nearly to level of umbilicus. No gong sound was obtainable over the abdomen. She was ordered one grain of opium in pill every six hours, and to be fed by nutrient enemata only.

On the next day the temperature fell to 99°, and the pulse rate to 78 per minute. She appeared better, and there was no abdominal pain. She was allowed liquid food by the mouth. Apparent improvement continued, but on the 6th

December—a week after admission—the left parotid gland became swollen, tense, and painful; a day or two later the right parotid gland was also affected, but to a less degree, and by the 11th December all parotid pain and swelling had ceased.

On the 9th December the patient complained of cramping pain in the left calf; the next day the foot was swollen, and it was evident there was thrombosis of the deep veins of the leg. After this the left thigh became swelled, and although all abdominal symptoms passed off, except an unduly violent throbbing of the abdominal aorta, she began to look ill, with sunken eyes, dry tongue, and a feeble pulse of 120 per minute.

On the night of the 18th December she had an attack of sudden pain in the abdomen, requiring morphia hypodermically for its relief; and on the 19th December, at 10.30 A.M., she suddenly became much worse, had a shivering fit with extreme feebleness of pulse, great restlessness, then unconsciousness, and died at 1 P.M.

No rigors or shivers occurred, except the slight shiver on the day of death; and the temperature was never over 99·2°, except on November 30, the day after the onset of the illness, when it was 103°, and on the 9th December, when it rose to 101·4°, coincidently with the thrombosis of the veins of the leg.

At the necropsy, performed on the 20th December by Dr. Dodgson, the inferior vena cava, the left common and external iliac, and the femoral and tibial veins of the left side were found completely occluded by soft ante-mortem clot; upwards the thrombosis extended into the right side of the heart, and had moulded itself within the commencement of the pulmonary artery. The clot appeared firmest and most organised in the left common iliac vein, but it was recent and soft even here. The viscera generally, including the stomach, the uterus and appendages, and the vermiform appendix, were absolutely healthy.

Remarks.—The initial symptoms of the patient's attack came on several days before those of thrombosis of the veins of the leg, indicating that the clotting began in the veins within the abdominal cavity,—the exact spot it was impossible to determine. The clot was perhaps a little more organised in the left common iliac vein than elsewhere, but judging from the sudden onset, with acute pain and tenderness in the epigastrium, the tension of the rectus muscle in this region,

and the amount of shock, the clotting began in the upper part of the inferior vena cava. Once begun, wherever it started, the clot must have extended downwards as well as upwards, till it caused death by intra-cardiac thrombosis.

No clue could be found to explain the occurrence of the thrombosis; except for a moderate degree of anæmia there was no constitutional state to account for it, and there was no local visceral disease of any sort.

Thrombosis, however, frequently occurs from causes which, with our present knowledge, are inexplicable; and this appears to be an example of what has been described as "primary infective thrombosis."

The only case I know of at all resembling it is recorded by Dr. Dowse in the *Lancet*, vol. ii, 1879, p. 268. A lady of forty-three years was suddenly seized with vomiting, shivering, and a feeling of faintness, followed on the next day by evidences of thrombosis of the femoral veins. After eleven days, on which repeated rigors occurred, she died, and the autopsy showed thrombosis of the veins of the lower limb and of the iliac veins. No cause could be found for the initial thrombosis, but the reporter of the case suggests it was due to shock and alarm at a thunderstorm raging at the time of the first symptom.

A point of note in the present case was that, although the vena cava inferior was occluded in its whole length, œdema only occurred in the lower limb of the left side, where it was accounted for by the thrombosis in the crural veins themselves. And it is perhaps worthy of remark, that the statement found in many text-books that parotitis is distinguishable from mumps by being unilateral and suppurative, is refuted by this and many other recorded cases.

But the chief clinical interest in the case is the resemblance of the symptoms to those of perforation of gastric ulcer.

The history of indigestion with epigastric pain, in an anæmic servant, culminating in an attack of sudden severe pain in the upper part of the abdomen, with collapse and subsequent tympanites, local tenderness, and pyrexia, certainly accorded with this diagnosis, for which she was sent to hospital.

There was, however, no ascertainable diminution in the area of liver dulness, and no bell sound was obtainable over the abdomen; in addition, the stomach percussion-note extended upwards as high as the fourth rib in the left axilla—evidence that the stomach was still distended.

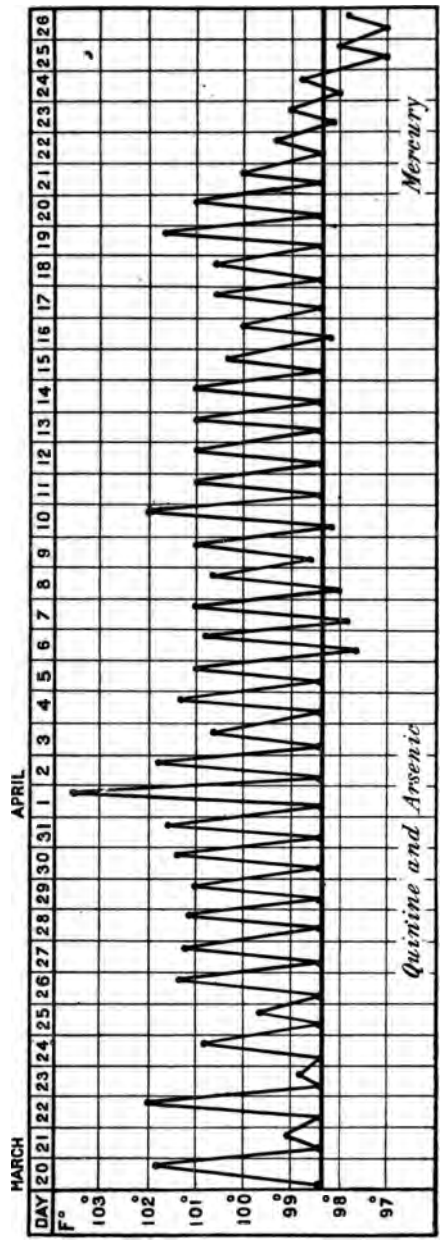
In the many acute abdominal conditions which simulate perforating gastric ulcer, the stomach is over-distended apparently from inhibitory paralysis of its muscular fibres; in perforation with escape of stomach contents it cannot remain long so over-distended, though no doubt it may do so where adhesions are present and the puncture minute. And though I do not suggest that signs of distension of stomach absolutely negative perforation, I think when they are present there is a presumption against perforation, and that this should be taken into consideration in deciding on immediate operation in doubtful cases.

V.—*A case of late Syphilitic Pyrexia* (*Intermittent Essential Fever of Syphilis*). By CAMPBELL WILLIAMS. *Read November* 23, 1900.

THE following case presents points of interest; firstly, because of the length of time intervening between infection and its appearance; secondly, in view of the prolonged mercurial treatment to which the patient had submitted, together with absence of all syphilitic manifestations for a year and a half; and lastly, in that it adds additional proof to the uselessness of antiperiodic remedies in these cases which react immediately to antisyphilitic treatment.

X., æt. 43 years, consulted me on 10th January, 1897, for a typical indurated chancre of the left side of the penile furrow. His inguinal lymphatic glands were but slightly enlarged. The dorsal lymphatic duct of the penis was not tangibly thickened. He was at once put upon mercury. The ordinary signs of syphilis ensued until June, 1898. The last of these was his tongue, which had been very troublesome, owing to severe ulceration of the dorsum, due to an ill-fitting gold plate. When he consented to replace this by a smooth vulcanite denture the tongue rapidly healed, and in June, 1898, he was free of all active signs of syphilis, and remained so until he discontinued treatment on 8th January, 1900. During this period of three years he had been practically treated continuously with mercury, alternating with iodides. For the last eighteen months of his course he did not show a symptom of active syphilis. The prolonged treatment was urged in view of his age at contracting the disease, as well as owing to the behaviour of his tongue, by ulcerating deeply in the early stage of the disease.

Towards the end of January, 1900, about a fortnight after his last visit, I learned that he had fallen a victim to the prevailing influenza, and was laid up. After this I heard nothing of the patient until 14th April, when he wrote to me saying that he was "very ill from post-influenzal intermittent fever," which had c[illegible] some weeks after the influenza." He gave a graphic [illegible] of his s[illegible] and temperature, which rose [illegible] on [illegible] and fell *to*


MARCH
APRIL
DAY
20 21 22 23 24 25 26 27 28 29 30 31 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26
F°
103°
102°
101°
100°
99°
98°
97°
Quinine and Arsenic
Mercury

normal by next morning. He noticed the "crises" in the early hours of the morning. Combined with this he complained of severe nocturnal pain in the right collar-bone; also that he was being treated with quinine and arsenic, but that they had no effect upon him, and that he was going steadily down hill. I advised him to come to town. This he did on 18th April, when I verified my suspicions as to the cause of his trouble, being confirmed in my diagnosis by his chart and his right clavicle, which was the subject of syphilitic periostitis. He was put upon mercury, which he commenced to take on 19th April, with the result that by the 25th April his temperature disappeared, and has remained normal ever since. The periostitis rapidly resolved, and the patient has regained his strength, weight, and colour, and feels as well as ever once more.

Recapitulation.—Febrile symptoms arose three years and two months after primary infection. Patient had been treated three years, practically continuously, with mercury and iodides, and for one and a half years of this period was free from any objective or subjective symptoms of syphilis. Mercury brought the temperature down to subnormal in six days.

VI.—*A case in which Splenectomy was performed for a Wandering Spleen.* By J. Bland-Sutton. *Read November* 23, 1900.

A MARRIED woman, æt. 34, was placed under my care, on account of a very movable abdominal swelling, which was regarded as a wandering spleen. The patient was the mother of seven children, and at the time of her admission into the Chelsea Hospital for Women considered herself to be two months pregnant. The belly wall, being extremely lax, permitted an easy examination of the viscera. When lying in bed nothing abnormal was obvious, but the moment she sat up in bed, or stood, then an elliptical mass appeared in the left lumbar and umbilical regions, and gradually descended to the iliac fossa. This body exhibited the outline of an enlarged spleen; the notches were not very obvious, but its shape, and the manner in which it could be moved over the intestines, convinced me that it was a wandering spleen. The dragging of this viscus on its pedicle produced a good deal of pain, and interfered so much with the patient's home duties that she was anxious for relief.

Careful observations were carried out on the blood by Dr. Victor Bonney, as to the numerical relation of the red and white corpuscles. When we were satisfied from repeated observations that the leucocytes were not in excess, I decided to remove the spleen. This was carried out May 28, 1900, through an incision in the left linea semilunaris.

The spleen, three times its normal size, was found in the left iliac fossa, swinging at the end of a thin, elongated pedicle, and rotated through half a circle. The vessels were secured by thin silk ligatures close up to the spleen (five in all were required), and the organ cut away. The pancreas was not involved in the pedicle, and after a careful but unavailing search for a spleniculus, the wound was closed in triple layers, silk for the peritoneum, fine silkworm gut for the muscles and fascia, and continuous silk for the skin.

The subsequent course of the case requires no comment, as convalescence was uneventful and rapid.

At the present time I have seven patients living from

whom I excised the spleen. These women report themselves from time to time, and none present any variation in health, or offer any indication that they are spleenless.

I thought it would be useful to append to this paper a short reference to each case, and this may satisfy some surgeons who are not assured as to the remote effects of splenectomy.

Case 1.—A woman, æt. 22, mother of one child. Splenectomy July, 1892, for axial rotation of a wandering spleen. For three years following the operation this patient reported herself, and permitted examination of the blood. Nothing noteworthy was detected. In 1895 she had an attack of right-sided pleurisy, and has suffered from menorrhagia; but this was satisfactorily explained by the condition of the uterus, namely, acute flexion and thickened endometrium. The flexion of the uterus was caused in the first place by the heavy spleen tumbling upon it.

In 1898 she was in good health. (*Clin. Soc. Trans.*, vol. xxvi, p. 46.)

Case 2.—Girl, æt. 17 years. Splenectomy performed in August, 1895, for an enlarged spleen, which dragged on the stomach, and caused recurrent attacks of jaundice. No alteration in the proportion of blood corpuscles. Patient reported herself in robust health early in 1900. (*Lancet*, 1895, vol. ii, p. 974.)

Case 3.—Girl, æt. 5 years. Splenectomy performed in August, 1895, for enlarged spleen associated with marked anæmia, but the corpuscles of the blood preserved their usual proportion to each other.

The anæmia quickly disappeared, and the little girl became strong and well.

September 21, 1900, the patient was reported to be "very well, looks healthy, is of an average height, but rather thin." (*Lancet*, 1895, vol. ii, p. 974.)

Case 4.—Woman, æt. 33, mother of four children. Splenectomy August, 1895, for a wandering spleen. A piece of the tail of the pancreas was also removed.

Last report, September, 1900. Patient in excellent health, and instead of being a "thin, spare woman," is "quite plump." (*Lancet*, 1895, vol. ii, p. 974.)

CASE 5.—Woman, æt. 35. Splenectomy performed in May, 1897. Spleen lacerated during an operation, when it bled so freely that it had to be excised.

Patient in good health two years later. (*Clinical Journal,* 1897, vol. x, p. 369.)

CASE 6.—Woman, æt. 21, mother of one child. Splenectomy September, 1897, for a tuberculous and wandering spleen. She had a quiescent tuberculous cavity in the lung.

September, 1900. Nothing to indicate that she was spleenless. (*Practitioner,* 1898, p. 288.)

CASE 7.—Woman, æt. 34, mother of seven children. Splenectomy performed May, 1900, for wandering spleen.

Patient was delivered of her baby in December, and in January, 1901, reported herself in excellent health.

VII.—*A case of Nephritis without Albuminuria; death in six weeks; no scarlatina; autopsy.* By W. P. Herringham, M.D. *Read November* 23, 1900.

JAMES G., æt. 3 years 10 months, was admitted into the Children's Hospital, Paddington Green, on March 29, 1899.

History.—Was run over on February 14; was admitted to a London hospital, and was in the ward for a fortnight. Is reported to have had "broken ribs." On the day of his discharge he had abdominal pains, and three days later sickness and offensive diarrhœa. These have continued till now. He has lost flesh. No history of scarlatina.

On admission.—Rather thin. Lungs and heart natural. Abdomen swollen and rigid. Tender and painful about navel. No enlargement of liver or of spleen. Slight rickets.

He was thought at first to have tubercular peritonitis, but he improved, and the diagnosis was not confirmed. The temperature was very irregular during the first week, but later became steadier, and lay between 98° F. and 99·5° F. The urine was thrice examined, and never contained albumen. It was passed in natural amounts.

He was sent down to the convalescent home belonging to the hospital on May 5, practically well, and remained there in good health for over two months. The home is visited weekly by the resident medical officers, and the children's temperatures are taken daily.

The first symptom of illness was on July 14, when he had marked œdema of the feet, which on August 1 spread to the legs, face, and eyelids. The urine was at once tested, but no albumen could be found.

He was brought back to the hospital on August 7.

On re-admission.—Great œdema of feet, legs, and face. The organs natural as before to physical examination. The urine very scanty, acid, containing no albumen and no casts.

From that time till his death on August 29 no great change took place. The dropsy was persistent, the urine very scanty—only about two fluid ounces per diem,—and the temperature rather raised, lying between 98° F. in the morning, and 100° F. in the evening. The bowels were regular.

Towards the end of his life the vomiting increased and the pulse grew weaker. Some diarrhœa appeared in the last week.

The urine was repeatedly examined, but no albumen was ever detected chemically, and no formed elements were ever found with the microscope.

At the *post-mortem,* all the organs, including the kidneys, appeared quite healthy to the naked eye.

Microscopic sections of the kidney and liver were made by Mr. Turner, Pathologist to the Hospital, and given by him to me. They present the following appearances:

The tubes in the labyrinth are all abnormal. They have a generally blurred aspect, the nuclei are in places absent, the lumen is blocked with material like that of the cells. Nearly all the tubes in the medullary rays are normal, but a few, which appear to be collecting tubes, are stuffed with similar material.

The glomeruli contain a granular structureless exudation. Thirty consecutive glomeruli were alike in this. There is no proliferation of the capsular epithelium, but there is perhaps some such change in the capillary tuft. The renal capillaries and arterioles are unaltered. There is no fibrosis, nor any small-celled infiltration.

The changes are, therefore, purely parenchymatous, affecting the labyrinth or secreting structure only. The disease cannot well be of longer standing than six weeks. It is not accompanied by congestion of the blood-vessels, yet the course is that of acute nephritis. Scarlatina can be excluded. There was some fatty infiltration in the liver.

Although most systematic writers mention these cases (*e. g.* Bristowe, 4th ed., 1882, p. 810; Fagge, 1886, vol. ii, p. 442; Osler, 1892, p. 743), they all speak of them as rare, and none of them quote any instances. Sir Dyce Duckworth wrote a paper in 1883 (*St. Bartholomew's Hospital Reports,* vol. xix, p. 321) on "Scarlatinal Dropsy with little or no Albuminuria," and after giving many references to other writers he concludes, "I have met with several cases in young children and adults in which there was extreme anasarca and no albuminuria, neither was there any history or likelihood of previous scarlet fever, and no visceral disease of any kind detectable, save enlarged lymphatics. These cases all recovered. But the anasarca was the solitary obvious symptom, and it seemed fair to exclude the idea of acute nephritis. . . . The only instances I have been able to find where

no albumen was present under these conditions are those of scarlatinal nephritis."

I myself have been unable to find any instance in literature since Sir Dyce Duckworth wrote, except as a consequence of scarlatina, and I therefore think that the present account is worth preserving as an explanation of a certain number of very doubtful cases.

Looking at the kidney under the microscope, I fail to see any reason why albuminuria should not have been present. The exudation in the glomeruli and in the convoluted tubes is almost certainly serum albumen, and it is just the case in which I should have expected to find a large amount in the urine.

The only two suggestions that occur to me are: that perhaps the albumen may either originally have been chemically different, or may have undergone some secondary change from the usual form, which may have rendered it irresponsive to the ordinary tests—boiling with acetic acid, nitric acid, and picric acid, which were employed; or that perhaps the exudation in the tubes and tufts was so great that it formed in itself a filter close enough to prevent the passage of albumen.

VIII.—*A case of Ankylostomiasis.* By W. J. Tyson, M.D. *Read December* 14, 1900.

A. M., æt. 34, has lived in China for fifteen years, and had been quite well till May, 1899 (except for occasional attacks of gout). He was told that he was suffering with malaria, and was ordered arsenic. At this time he was living at Singapore. In June he went to Hong Kong, and here the diagnosis of malaria was again made, and he was advised to go to England.

He stopped at Singapore on his way, stayed for fourteen days, during which time he became very anæmic, suffered from indigestion and flatulency, but was not sick. He noticed that his stools were often reddish in colour. Attacks of faintness were common, with much breathlessness and palpitation on the slightest exertion. He also noticed dimness of vision, with partial loss of hearing. He slept badly, and his appetite was indifferent.

During the sojourn of fourteen days in Singapore the malarial parasite was discovered in the patient's blood, and on account of the excessive anæmia the fæces were examined, and there was found in them the ova of the *Ankylostomum duodenale.* As he had arranged to leave Singapore, a course of thymol was advised to be carried out on board ship.

After fasting for one day and opening well the bowels, 10 grains of thymol were administered every four hours for three times, but besides making him sick no effect followed, the examination of the fæces giving no result.

Dr. Patrick Manson saw him on his arrival in London, and confirmed the diagnosis of ankylostomiasis and advised a course of thymol, and kindly placed him under my care. He was admitted into the Victoria Hospital, Folkestone, on September 23 last year. The notes taken at this time are the following:

He is a well-nourished man of good physique, weighing 13 st.; is evidently suffering from a severe form of anæmia by the extreme pallor of his skin and mucous membranes. Heart beats in the normal position, but there is a hæmic murmur at the base. There is no enlargement of the liver

or of the spleen; no œdema present, no retinal hæmorrhages. The urine has a sp. gr. 1020; no albumen or sugar present. He complains of pain in the epigastrium and insomnia. His appetite is spare, and this has never been ravenous or perverted at any time. His bowels are regular and he has no colic. His temperature on admission was 100° F., but fell to normal on the following day, remaining so while he was under observation.

The following treatment was adopted on September 24. Two doses of Carlsbad salts were given in the morning, and, when the bowels had acted, thymol was given as follows:

10.45 A.M.,	1st dose of thymol,	gr. xxx in	10-grm.
12.45 P.M.,	2nd „	„	cachets.
2.45 „	3rd „	„	
4.45 „	4th „	„	

The thymol made him feel rather sick, but had otherwise no unpleasant effect. During its administration the bowels acted several times. At 7 P.M. a further dose of Carlsbad was given and the bowels again acted. The fæces were carefully examined the same evening, and a large number of worms as well as ova were found in them.

September 25.—He passed a comfortable night, and feels better. The bowels acted once. Was allowed some solid food.

September 27.—He got up.

September 28.—He went home.

A week later the above treatment was repeated on account of two or three worms being found in the motions.

November 11.—He is now well and strong.

Blood Examination.

Before treatment	Sept. 18, 1899,	by Dr. Manson.	Red corpuscles	2·225,000
				2·400,000 p.c.mm.
	„ 25, „	by J. W. Stainer.	„	3·060,000 „
After treatment	„ 30, „	„	„	3·020,000 „
	Oct. 18, „	„	„	4·780,000 „
	Nov. 12, „	„	„	4·550,000 „

Patient's Weight.

1899.	August 30	180 lbs.	Before treatment.
	September 11 . . .	189 „	
	„ 30 . . .	184 „	After treatment.
	October 19 . . .	192 „	
	November 11 . . .	195¼ „	

Remarks.—Perhaps the only reason for recording the foregoing case is that it may lead some to think of the presence of this parasite as a cause of progressive and obstinate anæmia; and I think that it is quite possible this form of anæmia may be sometimes overlooked in England, especially when we consider how many more people travel to the East now than in former years.

The discovery of the worm was made as far back as 1838, but much attention was not paid to it until the outbreak of the notorious epidemic of anæmia among the workmen employed in making the St. Gothard Tunnel in 1880, and this was found to be due to the above parasite.

It was in this year that the specific treatment by thymol in large doses was carried out by Bozzolo, and has since been in constant and more or less successful use.

It will be observed that diminution in the number of the red corpuscles—one million or more per cubic millemetre—is the main alteration in the blood.

The patient when first seen was in a very anæmic and weakened condition. He could scarcely walk, and looked exceedingly ill.

The large doses of thymol had an extraordinary effect on the parasite, but apparently did not affect the patient in any poisonous way; but it must be remembered that at times serious symptoms, such as delirium, vertigo, the passing of dark brown urine, do follow the administration of the drug, and that death has sometimes ensued. The use of alcohol in any form must be forbidden, either before or after the giving of thymol, because of the ready solubility of the drug in it.

Whether the anæmia is brought about by the loss of blood by means of the worms, or whether it results from the dyspepsia, or whether the parasite produces a poisonous excretion, it is difficult to say; possibly all play some part in the production of the anæmia.

The description of the worm, and the pathological condition of the body which arises from its presence in large numbers, I need not deal with—they are well known.

It is interesting to note in this case the presence of the malarial parasite with the *Ankylostomum duodenale;* it is not uncommon for the two parasites to dwell in one host. When this is so it becomes important to settle the relative value of each cause; for the administration of thymol is not a suitable treatment for malarial disease, or any other form of anæmia.

The entire circumstances of the case, with an examination

of the blood and the fæces, must be considered, and a careful exclusion of such causes of anæmia as malarial disease, Bright's disease, cancer, tuberculosis, pernicious anæmia, etc., made.

IX.—*A case of Hæmatoporphyrinuria not due to Sulphonal.* By JAMES CALVERT, M.D. *With a Report on the Urine by* A. E. GARROD, M.D. *Read December* 14, 1900.

S. G., æt. 32, single, general servant, was admitted on January 15, 1900, into Elizabeth Ward, St. Bartholomew's Hospital. I was on duty at the time for Sir Lauder Brunton, who very kindly has given me permission to publish the case.

She has had no previous serious illness, but her general health has not been strong. She says she has "always" suffered from anæmia, and at times, though not lately, from indigestion. Her mother died from "ulcer of the stomach." On the 13th January, as she was doing her usual work she felt a pain in the epigastric region, became faint, vomited a large amount of blood, and then lost consciousness for a short time.

On admission she was very pale, her heart was somewhat dilated, a systolic murmur was heard over all the orifices, but loudest in the pulmonary region. Lungs natural. Abdomen:—stomach resonance natural, no pain nor tenderness on gentle pressure in epigastric region. Scybala felt in left iliac fossa. A little drowsy and apathetic. The diagnosis was gastric ulcer.

January 16.—As no urine had been passed for twenty-four hours—since admission,—a catheter was used, and 20 oz. of dark cherry-coloured urine drawn off. This urine was faintly acid, sp. gr. 1016; no albumen, no sugar, no blood, no bile, no casts. The spectroscope showed the bands of hæmatoporphyrin.

The patient noticed that her urine was much darker on the 14th January; it was "all right before then." Moreover, she says she passed similar dark urine about a year ago; at that time she was suffering from indigestion, but she had no hæmatemesis, nor, so far as she knows, any melæna.

She denies taking any sulphonal, and her doctor writes to say that he has never given her the drug. An enema brought away a considerable quantity of black blood.

January 19.—Urine still of the same character. Some uric acid crystals.

An examination of her blood was made:—hæmoglobin 48 per cent. Red blood-corpuscles = 2,292,000. White blood-corpuscles = 8500.

January 23.—Another blood examination was made:—hæmoglobin 54 per cent. Red blood-corpuscles 3,880,000. White blood-corpuscles 11,000.

January 26.—Another blood examination was made:—hæmoglobin 62 per cent. Red blood-corpuscles 3,968,000. White blood-corpuscles 11,000.

January 27.—Patient complains of weakness of both arms, and a sensation of numbness down the outer side of both arms. Urine still dark, though lighter than when she was first admitted.

January 28.—The menstrual period is just over; it lasted three days; scanty.

February 2.—During the last few days the urine has become much lighter in colour. On boiling it turns orange-coloured.

February 9.—Urine continues to get lighter in colour. Blood examination:—hæmoglobin 68 per cent. Red blood-corpuscles 4,144,000. White blood-corpuscles 9000.

February 22.—Urine no longer contains hæmatoporphyrin in abnormal quantity.

Treatment.—Until 31st January she had milk and lime water, peptonised milk, eggs. No medicine.

On February 1 she was put on Ferri Carbonas Saccharata, and the diet was increased in the usual manner.

Throughout the case the hæmatoporphyrinuria did not appear to give any manifestations of its own; all the symptoms present could be attributed to the hæmatemesis. The patient made an uninterrupted recovery, and left the hospital on the 8th March.

Remarks.—Eight cases have been recorded of hæmatoporphyrinuria not due to sulphonal poisoning. They have been enumerated in Dr. A. E. Garrod's article in the fourth volume of the *Encyclopædia Medica.*

They all ran a favourable course with the exception of Ranking and Pardington's two cases, which occurred at the same time in different rooms of the same house. These two cases were severe, presenting symptoms similar to those due to sulphonal poisoning; which, however, was carefully excluded. One of them died, the other recovered. There was no post-mortem on the fatal case.

The fact that the hæmatoporphyrinuria occurred after hæmatemesis is interesting, because Stokvis (*Schmidt's Jahrb.*, Bd. ccxxviii) holds that hæmatoporphyrinuria occurring in sulphonal poisoning is due to the absorption of altered blood, from hæmorrhages produced by the drug in the mucous membrane of the stomach and intestines—a view which is opposed, amongst other things, by the circumstance that such hæmorrhages are by no means constantly found at the post-mortems of these cases.

The blood-counts are instructive, showing as they do the increase of hæmoglobin and of red blood-corpuscles occurring together with the hæmatoporphyrin in the urine.

Dr. Garrod's Report on the Urine.

The urine of this patient closely resembled in its general characters that passed by patients with hæmatoporphyrinuria due to sulphonal. The specimen drawn off soon after admission was almost black, and the later specimens showed various shades of brownish red.

Its peculiarities of pigmentation may be grouped under three heads:

1. Hæmatoporphyrin was present in large amount, comparable to the quantities found in sulphonal cases, and far in excess of those met with in ordinary morbid urines.

2. The dark colour was hardly at all due to this pigment, but depended mainly upon the presence of one or more unknown pigments. This is a constant feature of the hæmatoporphyrinuria due to sulphonal, as Hammarsten first pointed out.

3. Some at least of the known pigments present showed anomalous features.

(1) When the urine was examined directly with the spectroscope, without the addition of any reagent, the blue end of the spectrum was seen to be much obscured. The narrow band in red of the so-called "alkaline" hæmatoporphyrin spectrum was fairly distinct, but, as is often the case in sulphonal specimens, the bulk of this pigment was obviously present in the form in which it yields the so-called "metallic" spectrum, which consists of two bands, which somewhat resemble those of oxyhæmoglobin.

Acetic ether, which usually extracts hæmatoporphyrin readily from urine acidified with acetic acid, in this instance hardly took up any of the pigment; but the soda method, which consists in the precipitation of hæmatoporphyrin upon

the phosphates, by the addition of 20 c.c. of a 10 per cent. solution of sodium hydrate for each 100 c.c. of urine, threw down practically all the hæmatoporphyrin present. This method, which succeeds admirably with normal and ordinary morbid urines, has failed to give satisfactory results with the sulphonal urines upon which I have tried it.

On extracting the washed phosphate precipitate with alcohol acidified with hydrochloric acid, a rich pink solution of acid-hæmatoporphyrin was obtained. The pigment was also precipitated by adding glacial acetic acid to the urine (5 c.c. to each 100 c.c. of urine), according to Nebelthau's method.

(2) The fact that the hæmatoporphyrin could be completely removed by the soda method greatly facilitated the isolation of other abnormal pigments. The alkaline filtrate regained the original tint of the urine when re-acidified with acetic acid, but now showed no hæmatoporphyrin bands. To a portion of this filtrate solid barium chloride was added, short of complete precipitation, and in this way a pink precipitate was obtained, which was repeatedly washed with water until the washings were colourless. The washed precipitate was then treated with dilute sulphuric acid, and a dark purple solution was obtained, which was filtered from the precipitate of barium sulphate.

From this solution none of the various solvents which were tried, such as chloroform, ether, acetic ether, and amylic alcohol, took up the purple pigment, even after neutralisation of the excess of acid. Evaporation of the neutral solution upon a water-bath partly destroyed the pigment, and after one or two evaporations its solutions had a sepia instead of a purple colour.

An alcoholic solution was obtained by saturation with ammonium sulphate, and shaking the saturated liquid with absolute alcohol, as in the process for the extraction of urochrome from urine. The alcohol, separating as a supernatant layer, carried up the purple pigment with it.

The impure alcohol solution thus obtained had a rich purple colour. It showed no definite absorption band in any part of the spectrum, but there was a shading in the middle portion of the spectrum too ill-defined for measurement.

Alkalies almost decolourised the pigment, leaving the solutions of a pale neutral tint, but re-acidification restored the purple colour.

Aldehyde had no obvious effect upon the alcoholic solution. Peroxide of hydrogen gave to it a more pink tint.

The purple pigment was found to be readily soluble in water and in acetic ether; not at all readily in chloroform, amyl alcohol, or absolute ethylic alcohol.

This purple pigment has not, as far as I can ascertain, been previously described. That which most closely resembled it was a reddish-purple colouring matter met with by Leube, which, however, differed in being readily extracted from the urine which contained it by merely shaking it with ether. It also differed in its behaviour with various reagents.

(3) As the case went on the urine gradually resumed the ordinary character, but the excretion of considerable amounts of hæmatoporphyrin outlasted the presence of the purple pigment.

As is not unfrequently the case with sulphonal urines, the hæmatoporphyrin, after extraction, exhibited peculiarities of behaviour. For example, I failed entirely to get it into solution in chloroform, even from an aqueous-alcoholic solution acidified with acetic acid, from which the pigment is usually readily extracted by this solvent; but from a similar extract which had been kept for some months chloroform took up the pigment readily.

The later and paler specimens of urine showed a dark urobilin band, but this differed from ordinary urobilin in being less defined towards the red; and the ordinary processes for the extraction of urobilin, such as the ether-chloroform method, failed to extract urobilin in any quantity. The pale urine passed when the attack was passing off showed a further peculiarity, viz. that when it was simply boiled it assumed a rich orange colour, and the urobilin band gained *greatly* in intensity, and became more sharply defined. This is a feature which I have never met with in any other urine.

The description of the unstable purple pigment is necessarily very incomplete, chiefly on account of my failure to obtain it in a pure condition without change; but it will be interesting to see whether this same pigment can be extracted from sulphonal urines. That it is present in them I am inclined to think, from the behaviour of the barium precipitates obtained when Salkowski's method of extraction is applied to such urines.

There could be no doubt that in this case the dark colour of the urine in the early stages was almost wholly due to its abundant presence.

X.—*A case of Pneumococcal Suppurating Pericarditis treated by incision and drainage: right lobar pneumonia: right empyema: death.* By JOHN FAWCETT, M.D., and F. J. STEWARD, M.S. *Read December* 14, 1900.

E. W., æt. 8 years, was admitted into Guy's Hospital on July 16.

The child had always been delicate, but never seriously ill previously.

The present illness commenced on July 10, with pain in the right side followed by delirium.

On admission.—The boy was very thin and of feeble development. He had signs of lobar pneumonia at the right base.

July 17.—Some little increase of præcordial dulness was noted, and a soft "to and fro" bruit localised to an area the size of a shilling, below and internal to the left nipple.

July 19.—The temperature fell almost to normal, and the signs at the right base were disappearing. Between this date and July 27 the temperature varied between 98° and 100° F., only reaching 101° on one occasion. The heart-sounds had a triple rhythm, and were becoming gradually more muffled.

July 27.—The præcordial dulness, which had gradually been increasing in extent, now reached the first intercostal space above, towards the right one inch beyond the right edge of the sternum, and towards the left as far as a vertical line two inches external to the left nipple.

The upper portion of the epigastrium had, during the last two or three days, become more prominent and was very tender on pressure. There was no œdema of the chest wall, nor any signs of compression of the lung over the left base posteriorly.

Although the area of præcordial dulness sounds from the description to have been much increased, it was not in reality very large, as the child's chest was so narrow and small.

By this date the cough had again become frequent and

severe, and the pulse and respiration rate had increased. The temperature, however, was little raised.

As the boy was becoming visibly thinner, and was taking his food, and sleeping, very badly, and as, also, I thought that the effusion, although not extensive, was very probably purulent, I asked Mr. Steward to explore with a needle; and pus having been found the pericardium was opened the same day. The details of this and the operation are given in Mr. Steward's notes of the case.

After-history.—The child's general condition improved very much. The cough became less troublesome, the pulse stronger, and he took his food, and slept, very much better. This improvement was maintained until August 9, although for the preceding four days his temperature had been more variable and pulse quicker.

August 10.—Evidence of a localised empyema was detected on the right side. This Mr. Steward incised and drained.

August 15.—The child had up till now again improved in his general condition, but on this date suddenly showed signs of collapse, and died in a few hours.

Typical diplococci were found in the pericardial pus. The empyema unfortunately was of streptococcal origin. Only one cultivation was made from each site.

Autopsy.—Pericardium:—The layers were in exact apposition all over. There was not any pus in the sac. The pericardium was somewhat thickened with a little recent lymph on its surface. The acute inflammatory change was obviously subsiding, and the appearances were those of obliteration of the cavity formed by the effusion.

On left side the pleura was adherent to the pericardium. There was a little recent capsulitis over both liver and spleen. The lower lobe of the right lung was tough and firm. The tubes were somewhat dilated. I thought there was evidence of early fibroid change. The other viscera were normal.

Remarks.—The interest of the case was chiefly in that, as the result of the autopsy showed, the inflammatory pericardial change was clearing up, and that had it not been that the boy developed a streptococcal empyema, I think it probable he would have survived.

He was an extremely thin and ill-developed boy, a most unfavourable subject for so severe a series of infections; yet he lived eighteen days after the operation, and a marked improvement in his general condition, with a gradual diminu-

tion in the discharge, was taking place until the empyema developed.

It is interesting to note the almost complete absence of friction-sound. It was only heard twice, and then its significance was somewhat doubtful. The boy was frequently examined for it both by the house physician and myself.

Another point of interest is the slight pyrexia that existed. There were no rigors, but the boy sweated very freely.

The cases so far recorded, including this case, which have been treated surgically, are forty-five in number, with sixteen recoveries and twenty-nine deaths; the most important recent papers on the subject being by Messrs. Shattuck and Porter (*Trans. Amer. Assoc.*, Philadelphia, 1897), and by J. B. Roberts (*American Journal*, 1897). They gave details of thirty-six cases; and in addition there are six by Ljunggren, one by Sorel and Dalons (*Echo Med.*, Toulouse, 1900, xiv, pp. 213—215), and one by Ogle and Allingham (*Lancet*, vol. i, 1900).

F. J. Steward, M.S.

I was asked by Dr. Fawcett to see this case on July 27, the eighteenth day of the illness. In view of the facts that the area of cardiac dulness had been steadily increasing for ten days, that it was now considerable in extent, and, moreover, that the boy had been steadily going downhill during this time, I agreed with Dr. Fawcett as to the advisability of exploring the pericardium. A needle was inserted in the fourth left space about one inch from the margin of the sternum, and thick yellow pus withdrawn. A few hours later the following operation was performed. The skin of the chest having been thoroughly cleansed in the usual manner, the A.C.E. mixture was administered, and taken well. An incision was made over the fifth left costal cartilage, the perichondrium stripped off, and about one inch of the cartilage removed. As no pleura came into view, and the pericardium was easily seen and felt, this was incised.

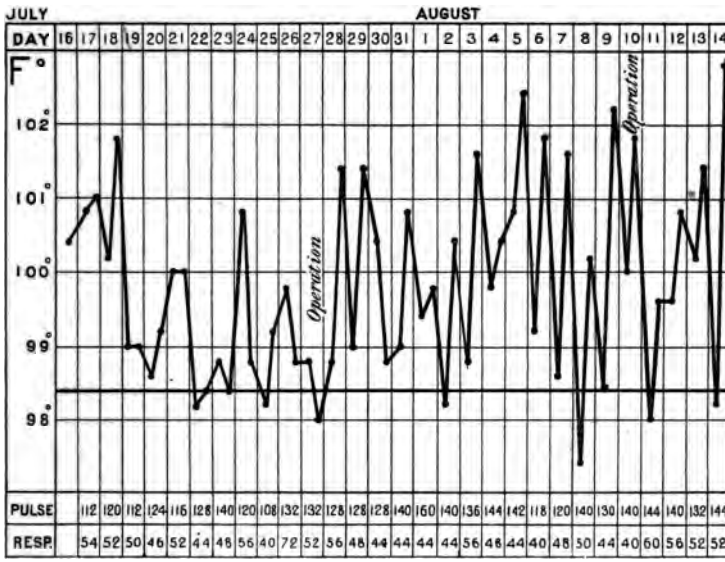
About eight ounces of thick pus having escaped, a short drainage-tube of large diameter was inserted, and dressings applied. The operation was quite simple, and in fact only occupied a very few minutes. During the first twenty-four hours a large quantity of pus drained away, necessitating frequent change of dressings. Subsequently the discharge gradually lessened in amount, and after the fifth day very

little came away. An attempt was made to keep the boy on his face in order to assist drainage. As, however, the position seemed very irksome to the patient, the attempt had to be abandoned.

Although drainage appeared to be quite satisfactory, and the pus remained quite sweet, reference to the chart will show that the temperature steadily rose after the operation, each day reaching a slightly higher level than the previous day.

On August 10 it was found that the pyrexia was accounted for by a small empyema at the right base. This was at once opened and drained, about four ounces of thin pus escaping. Death took place five days later.

Remarks.—Examination of the specimen clearly demonstrates once more that early incision and drainage is undoubtedly the correct treatment in these cases, although



the termination in this case was unfortunately fatal. For whereas the pericardium on July 27 was of considerable size, and must have contained at least ten ounces of pus, and probably more, it will be seen on examining the specimen, that when death took place—nineteen days after the operation—the cavity of the pericardium was practically obliterated.

The result also shows that drainage in this position, namely, through the fifth costal cartilage, is quite efficient, although it was not possible to keep the boy on his face, as would seem advisable.

Note.—Since this paper was read a more complete list of cases has been published by C. B. Porter (*Ann. of Surgery*, December, 1900). Here 51 cases are given. Including this case the total is therefore 52 cases, of which 20 recovered and 32 died.

XI.—*A case of Gonorrhœal Rheumatism, with Bursæ filled with Concretions of Urate of Soda.* By W. G. Spencer. *Read December* 14, 1900.

THIS case is recorded since, although an anomalous one, it may have some interest in connection with exceptional cases of gout.

A coloured clerk, æt. 33, came over to this country for treatment from the West Indies. Twelve or fourteen years ago he suffered from gonorrhœa, and about ten years ago had had a urethral stricture divided under anæsthesia, but from the time of the operation no instrument of any kind had been passed down the urethra. Dating from six or seven years ago, he had had spasmodic attacks of pain in the legs, lasting two or three weeks, especially in the months of August and September (*i. e.* in Demerara). During the attacks his knees were stiff; between the attacks he could walk and bicycle. Six years ago the present swelling formed on the dorsum of the left foot, those over the tubercle of the right tibia and on either side of the heel eight months ago. Sometimes they had been much swollen. He had never experienced any difficulty with micturition.

The patient had had no other disorder. He had only drunk whisky, and that not every day; he had not drunk rum. He had not had anything which could be considered a gouty attack. His father died aged seventy-seven. Two years before his death he had pains in his right wrist, which he used to wrap in flannel. With this exception there was no family history of gout whatever.

On arrival in this country the joints, especially the knees, were so inflamed that he could not walk. The skin over the knees was red, shiny, hot, and tender, and the joints could not be moved without pain. Of the other joints, the left ankle and dorsum of the foot were most affected. A bougie passed into the urethra discovered a stricture, largely spasmodic, situated at the junction of the membranous and bulbous urethra, and admitting at first only a No. 5 bougie.

So far the case was simply one of gonorrhœal rheumatism kept up by a spasmodic stricture. But besides this the patient showed four bursal enlargements, each

about ¾ inch in diameter. One was situated on the dorsum of the left foot, attached to the inner and middle cuneiform bones; two, one on each side of the left tendo Achillis, just above its attachment to the os calcis, and these communicated partially; the fourth was over the tubercle of the right tibia. It was supposed that these bursæ contained fluid, and were of the same nature as the joint affections. The patient was therefore treated for a fortnight by slow dilatation of the urethral stricture and the application of belladonna fomentations. Under this treatment the joints became rapidly less swollen, the pain subsided, and they could be moved more freely. But meanwhile this treatment had no effect upon the bursæ.

With the object of finding out whether these bursæ contained gonococci, they were explored with a syringe. A small amount of thick, white, semi-solid material was extracted, which our pathologist, Dr. Lazarus Barlow, found to be simply a mass of acicular crystals of sodium biurate without any organisms. The bursæ were then incised and their contents collected, in all about 3 drms., of pure white needle-shaped crystals of sodium biurate without any admixture. The walls of the bursæ were freely scraped, and afterwards dressed daily with gauze. Some urate of soda crystals could be found in the scanty discharge, but they all rapidly healed. Meanwhile he was given some colchicum.

The patient was able to leave for home after six weeks, the knees being cold and quite free from pain, and the bursæ healed. He was instructed to take colchicum at intervals and to pass a bougie.

In some way, not at all evident, these bursæ must have been connected with the gonorrhœal rheumatism, and the concretions of urate of soda must have been a product of the inflammation. The patient was clear in his statement that the bursæ swelled at the time the joints were attacked. On the dorsum of the right foot was a diffuse teno-synovitis, such as is often seen in gonorrhœal rheumatism; and this was absorbed as a result of the urethral dilatation and the belladonna fomentations; whilst the circumscribed bursa in the same situation on the left dorsum, containing urate of soda, remained unaltered, and required active surgical interference.

Yet there was nothing in the patient or his family history pointing to a tendency to gouty deposits as a result of inflammation.

XII.—*A case of Fatal Pancreatic Cyst of enormous size simulating Ascites.* By SIDNEY PHILLIPS, M.D. *Read January* 11, 1901.

H. C., æt. 22, farm labourer, enjoyed good health until August, 1899, when he received a kick from a horse in the upper part of the abdomen, and had to give up work for three weeks, the first two of which were passed in bed under medical treatment. The day after resuming work sudden acute abdominal pain laid him up another day, and from that time onwards he had had occasional colicky pains in the abdomen, and vomited once; he never had any hæmatemesis or melæna, and the bowels always acted regularly.

He came under my care in St. Mary's Hospital on January 2, 1900, complaining of pain and tenderness in the abdomen, which was much enlarged, measuring thirty-four inches at the level of the umbilicus; the abdominal wall was very tense, the umbilicus everted, and there was dulness on percussion all over the abdomen except in the region of the stomach; the percussion dulness did not alter freely with position; there was a well marked fluid thrill—the liver edge could not be felt owing to the abdominal distension. He was a pale thin man; the tongue was clean, appetite fair, bowels a little relaxed. Pulse 108 a minute, temperature normal. Urine normal, small in quantity. There was no œdema of the feet.

On January 9 there were signs of left pleural effusion verified by a hypodermic needle.

He got gradually weaker, and the girth of the abdomen increased, being thirty-five and a half inches on January 19.

On January 20 a Southey's tube introduced into the abdomen removed 145 ounces of clear slightly yellow fluid, of sp. gr. 1002. The abdomen soon became as tense as before, and at my request, on February 3, Mr. Pepper opened the abdomen just below the umbilicus. A very large amount of fluid was evacuated; the finger introduced into the wound impinged on soft fibrous adhesions readily broken down, and on each separation of these adhesions fresh loculated collections of fluid escaped.

A drainage-tube was left in the wound, which soon healed

up, but the patient was sick several times during the few days after the operation, and he gradually lost strength, and died on February 13.

The fluid which had escaped from the abdomen through the Southey's tube was clear, slightly tinged yellow, and to the naked eye exactly resembled ordinary ascitic fluid.

Post-mortem examination by Mr. Plimmer.—On reflecting the abdominal parietes the appearance presented was that of a large ascitic effusion encysted behind the great omentum, and extending down to within two inches of the symphysis pubis; below the effusion a few coils of collapsed small intestine were visible. The upper third was found to consist of the stomach greatly dilated. The lower two thirds consisted of a large pancreatic cyst; the walls of stomach and cyst so closely resembled each other, and were so closely adherent, that the line of demarcation was with difficulty recognised.

The pancreas was normal in appearance and size; adherent to the whole of its anterior surface, and communicating with the glandular substance of the anterior part of the head by a fistula one sixteenth of an inch in diameter, and half an inch long, was the above-described enormous cyst. It was bounded behind by the pancreas, to which it was densely adherent; it was also adherent to the duodenum and stomach, also to the spleen; the transverse colon was adherent to it below, and slightly posteriorly, so that it was not visible when the abdomen was opened. The great omentum was adherent to and almost incorporated with the lower half of the posterior wall of the cyst. The superior mesenteric vessels were adherent in their first few inches to the posterior wall.

The cyst contained about three litres of translucent greenish-yellow fluid; but as the nature of the accumulation was not at first recognised it was drained away, being thought to be ascitic fluid. The cyst wall varied from one sixteenth to three eighths inch in thickness, and was coated inside with material resembling coagulated lymph. The stomach was much dilated.

Remarks.—The cyst there seems little doubt arose from the injury the man received seven months before his death.

It was unusual in the enormous size the cyst attained, virtually filling up the whole abdominal cavity. It was indistinguishable during life from an ascitic collection, for though it was noticed that the percussion dulness altered but little with the position of the patient, this was thought due to

adhesions, and probably this was the case. Even at the post-mortem examination the nature of the case was only ascertainable after careful dissection, and I thought during life that the case was one of tubercular peritonitis.

The fluid from the abdomen exactly resembled in appearance ordinary ascitic fluid; it was examined for tubercle bacilli, but I regret not for the special indications of pancreatic secretion. As regards the treatment, neither tapping nor laparotomy had any effect on the accumulation of fluid, and seeing the extensive growth and many adhesions of the cyst wall, I do not think any treatment could have offered any hope of success.

XIII.—*A case of Subacute Combined Degeneration of the Spinal Cord in association with Pernicious Anæmia.* By FREDERICK E. BATTEN, M.D. *Read January* 11, 1901.

THE interest of the case about to be narrated lies in the fact that it is one of combined degeneration of the spinal cord, associated with a condition of blood that may be characterised as pernicious anæmia.

The association of spinal cord degeneration with profound anæmia was pointed out by Lichtheim in 1887, and since that time by the writings of numerous authors on the Continent, and by Bowman, Taylor, Clarke, and Russell in this country, the disease has obtained general recognition. Many of the cases have been described as pernicious anæmia, others simply as profound anæmia. In a series of cases, however, published by Russell, Collier, and myself, which presented the symptoms of combined degeneration of the cord, the blood examination did not show those changes which might be considered characteristic of pernicious anæmia.

It is rather with a view to show that an exactly similar pathological condition may occur with pernicious anæmia that this case is brought before the Society.

For the notes and for the opportunity of examining the case I am indebted to my friend Dr. Hugh Smith, and for other clinical details both to Dr. Harry Campbell and to Dr. Guthrie, under whose care the patient was prior to his admission to "Friedenheim."

The case is that of a man, æt. 45, who was admitted to the "Friedenheim" under the care of Dr. Lush on April 6, 1900, and he gave the following history.

About five or six years ago he noticed that he was getting pale, and four years ago he was very short of breath.

Three years ago he attended the North-West London Hospital, and was admitted as an in-patient under the care of Dr. Harry Campbell in February, 1897. He was then said to be suffering from pernicious anæmia, and in reply to a note which I wrote to Dr. Campbell, he regrets that the notes cannot be procured, but he says that the case presented all

the typical symptoms of the disease,—intense anæmia, lemon colour, dark urine, defective coloured corpuscles and hæmorrhages; there was considerable gastro-intestinal irritation, which was attributed to the arsenic which he had been taking. He remained in the hospital for three months, and after being discharged he attended as an out-patient for a further period of two months. After this he did some light work till seven months ago, *i. e.* about October, 1899, when he noticed cramps in his legs, at first at night, and then during the day, and on November 24, 1899, he was admitted to Regent's Park Hospital under the care of Dr. Guthrie, who has most kindly supplied me with the following notes of his condition while there.

The patient had well-marked spastic paraplegia, with rigidity and spasmodic jumpings and involuntary crossing of the legs, which he could not control; the left leg was most affected, and there was diminished tactile sensation over the inner side of that thigh. The knee-jerks were exaggerated, ankle-clonus was present, and Babinski's extensor response was well marked on the left side and present on the right. The patient complained of aching gnawing pain in both lower extremities from the hips to the knees. When first admitted he had almost no control over his bladder, but after injections of strychnine this improved until he could hold his water for two hours; strychnine had, however, to be given up, as it increased the spasm of the legs. He had two severe attacks of præcordial pain, with faintness and cyanosis. He had the typical lemon colour of pernicious anæmia. The optic discs were very pale, but no hæmorrhages were seen. His teeth were discoloured, but fairly good. He said that he had never suffered from toothache, and his digestion had always been good until the anæmia set in. He was discharged from the hospital on February 2, 1900, and from that date till April 6, when he was admitted to the Freidenheim, he seems to have been at home.

The following was the condition of the patient, taken partly from notes made by Dr. Smith shortly after admission, and partly from notes made by myself on the 21st of April, when I first saw him.

The patient was extremely anæmic, and of a yellow colour; there was no jaundice, but the conjunctivæ presented the yellow fat appearance; he was thin but not emaciated. There was a bedsore over the sacrum.

The mental condition of the patient was clear, and there

had been neither delirium nor delusions while he had been in the home.

The pupils reacted well to light, the ocular movements were good, and there was no nystagmus. The fundus was normal, and no retinal hæmorrhages were seen.

Movements.—All movements of the face and tongue were well performed.

All movements of the arms could be performed, and there was no more inco-ordination or intention tremor of the hands than could be accounted for by the extremely weak condition of the patient.

The legs could not be raised off the bed; there was a little power of flexion, the limbs were spastic, and frequently contracted involuntary.

Reflexes.—The arm-jerks were present, the abdominal reflexes were present. The knee-jerks were exaggerated, and there was double foot-clonus, the plantar reflex gave very little response, and not of the extensor type. (H. R. S.). The muscular irritability was preserved, and the skin reflex was present. There was some anæsthesia over the feet and legs below the knees, but on the thighs and abdomen he certainly felt well. No anæsthesia could be detected in the fingers.

The physical examination of the chest revealed nothing abnormal.

The liver was somewhat enlarged, but the spleen could not be felt; there was no enlargement of the lymphatic glands.

Dr. H. R. Smith at this time examined the blood, and stained the films by the Ehrlich-Biondi stain. Macrocytes, microcytes, poikilocytes, and large nucleated red corpuscles were present; there was a distinct increase in the lymphocytes.

On April 21 I saw the man for the first time. The condition was very much as above described; the knee-jerks were present but not active, the ankle-clonus which had been present was no longer obtainable, and the plantar reflex, although feeble, gave an extensor response.

The anæsthesia was as above described.

The lower limbs were still slightly rigid, but the muscles of the calf were flabby, and the anterior tibial group were wasted. A bedsore was present over the sacrum.

The blood was extremely pale, and the following account has been furnished by Dr. Phear, who most kindly undertook its examination.

Red corpuscles .	574000	to the c.mm.
White corpuscles .	8000	„ „

Differential count of white cells.

Polymorphonuclear cells .	32	per cent.
Lymphocytes . . .	65	„
Large hyaline cells . .	1	„
Coarsely granular cells .	1	„
Cells resembling myelocytes	1	„

The red corpuscles, of which the number was remarkably low, included distorted forms of every kind of shape and every degree of size; some of the red corpuscles were vacuolated, a few were nucleated. The character of the red cells was such as one would expect to find in a case of pernicious anæmia. There was no leucocytosis. The actual number of the polymorphonuclear cells to each c.mm. of blood was very low (about two-thirds of the normal); the number of lymphocytes was comparatively increased. This change in the ratio of polymorphonuclear cells to lymphocytes has been noticed in pernicious anæmia. It is not unusual to find a small number of myelocytes in pernicious anæmia (Plate IV).

On April 24 the bedsore had increased in size, and there had been some delirium at night.

On April 30 the following note was made:—"The strength has entirely failed, the patient lay in a typhoid state, taking no food. There was complete loss of control over the bladder and rectum. The knee-jerks could still be obtained, but were below normal, ankle-clonus was absent, and the plantar reflex was slight, and inclined to flexion rather than to extension. The legs were drawn up when the soles were stimulated He did not seem to feel the prick of a pin on the legs or abdomen."

The patient died on the following day.

The *post-mortem* was performed forty hours after death. Tissues all extremely dry and bloodless; there was some œdema of the surface of the brain, which otherwise appeared normal; nothing abnormal could be seen in the external surface or membranes of the spinal cord; on section, however, the posterior and lateral columns showed the grey translucent appearance characteristic of sclerosis. The lungs appeared normal, the walls of the heart were pale and friable. The liver was enlarged, of a pale red colour, and not obviously fatty, and on testing it for free iron a typical reaction could not be obtained.

The spleen was enlarged, soft and pulpy.

The kidneys were slightly granular, but showed no other change. The blood taken at the time of post-mortem showed the same changes as the specimen examined during life.

Microscopical examination.—All parts of the nervous system have been examined by the four following methods. Nissl's method for staining the cells, Weigert-Pal's for staining the medullated nerve-fibres, Marchi's for showing any recent degeneration in the medullate fibres, and van Gieson's method for the vessels and connective tissue.

Cortex.—The cells of the cortex appear normal, the tangential and other medullated fibres appear normal, and there is no recent degeneration in either the grey or white matter.

The most striking change is seen in sections of the cord stained by the Weigert-Pal method; no degeneration can be seen in the pons or upper part of the medulla; at the level of the posterior column nuclei there is well-marked degeneration of the funiculus gracilis (Plate I, fig. 1, *f. g.*), and to a lesser degree of the funiculus cuneatus (Plate I, fig. 1, *p. c.*); degeneration is also present in the direct cerebellar tracts (Plate I, fig. 1, *a. c. t.*); no degeneration is, however, visible in the pyramids. In the upper cervical region there is marked degeneration of the posterior columns and of the direct cerebellar tract, and also to a lesser degree of the pyramidal tracts, but the degeneration is not confined to the area occupied by these tracts, but extends into the antero-lateral tract. At the level of the fifth cervical (Plate I, fig. 2) there is marked degeneration of the posterior columns, leaving only a band of normal fibres bordering on the grey matter; the periphery of the cord in the region of the direct cerebellar tract is markedly affected, as well as a symmetrical area on either side of the ventral median fissure. At the level of the seventh cervical the same features are visible. In the dorsal region the sclerosis is more extensive, involving nearly the whole of the white matter, except an area lying lateral to the anterior horns, and the white matter in immediate contact with the grey matter (Plate I, fig. 3).

This description practically holds good for all sections taken from the dorsal region of the cord.

In the upper lumbar region there is degeneration in the posterior columns on either side of the posterior median fissure (Plate I, fig. 4), and also in the crossed pyramidal tracts; in the lower lumbar region the degeneration in the

posterior columns becomes less extensive, and vanishes altogether in the sacral region, only a very little degeneration being visible in the crossed pyramidal tracts.

By the Marchi method the degeneration is seen to have wider limits than is indicated by the Weigert-Pal method; a few degenerate fibres can be seen in the pyramidal tracts in the upper segments of the pons; a fairly well-marked band of degenerate fibres can be seen in the region of the ventral cerebellar tract passing upwards to the velum (Plate II, fig. 1).

In sections through the olive there is most extensive degeneration in the direct cerebellar tract, and scattered degenerate fibres throughout both pyramids (Plate II, fig. 1).

In other sections of the medulla and cord the degeneration, as shown by the Marchi method, occupies the same regions as have been above described, except that there were fewer recently degenerate fibres in the parts that showed most sclerosis, while they were more numerous around the margins of these sclerosed areas (Plate II, figs. 1—11).

A considerable amount of vacuolation is present in the dorsal region of the cord, becoming less as one approaches the cervical and lumbar regions. The anterior and posterior roots show no change either by the Marchi or Weigert-Pal method.

The cells of the anterior horns, both in the cervical and lumbar regions, are normal, both in number and appearance. In the dorsal region there appear to be rather a few cells, but those seen are well stained and normal. Many of the cells are pigmented. The cells of Clarke's column are normal.

In sections of the membranes stained by van Gieson's method, the membranes, arteries, and veins appear normal. In the most sclerotic areas of the cord the vessels are uniformly thickened, and there are marked perivascular spaces (Plate III), but there are no hæmorrhages. In the less sclerotic areas of the cord the vacuolation is well seen; in the normal areas of the cord the vessels appear normal. There is no inflammatory condition of the grey or white matter.

The liver was kindly examined for me by Dr. Fletcher. He found that free iron was present, but it did not give a very good reaction. When stained by Marchi there is a considerable amount of fat, situated mostly in the periphery of the lobule.

The heart showed a considerable amount of fatty change, but the striation of the fibres was well preserved.

The kidneys showed some interstitial change, but were otherwise healthy.

The vessels in the spleen appeared thickened.

Remarks.—The case above described exhibited symptoms that pointed to involvement both of the lateral and posterior columns, and although the course which the disease ran did not correspond in all details with the usual type of subacute degeneration of the cord, yet the symptoms were sufficiently typical to render the diagnosis during life fairly certain.

I had hoped to have given the Society some tables showing the relative frequency with which the disease was associated with pernicious anæmia, but on going through the published cases it seems almost impossible to compile a satisfactory table in which the symptoms and pathological conditions were those of combined degeneration, and the condition of the blood one of pernicious anæmia. The cases published by Von Noorden, Eisenlohr, Leyden, Bowman, Rothman and Lloyd, and some of those published by Minnich and Nonne, undoubtedly belong to the same type as the present case. On the other hand, many of those published by Nonne, and more especially the cases in his last paper, in which he examined the spinal cords of cases of pernicious anæmia, cannot be regarded in the same category as the present case. How far one will be able to separate cases of combined degeneration of the cord from cases of profound or pernicious anæmia with spinal cord change seems to me at present uncertain, and one cannot but suggest that as the examination of the nervous system of these cases, both clinically and pathologically becomes more systematic, it will be evident that they pass gradually from one to the other. For the present, at any rate, it is useful to distinguish clinically, as a separate group, those cases which exhibit symptoms of combined degeneration of the cord with profound anæmia, from cases of profound or pernicious anæmia with changes in the spinal cord.

What proportion of the cases will show a condition of blood that may be designated as pernicious anæmia must remain for the present doubtful; it is well to note, however, that Dana,* in a recent paper, states it to be as high as 10 per cent.

With regard to the pathology of the disease it is unnecessary here to enter into a detailed discussion, for that has

* Dana, *Journal of Nervous and Mental Diseases*, vol. xxvi, 1899, p. 15.

DESCRIPTION OF PLATE I.

Illustrating Dr. Batten's case of Subacute Combined Degeneration of the Spinal Cord.

Sections of the medulla, cervical, thoracic, and lumbar regions of the spinal cord stained by the Weigert-Pal method.

FIG. 1.—Section taken at the upper level of the pyramidal decussation, showing extensive degeneration in the funiculus gracilis (*f. g.*) and to a lesser degree in the funiculus cuneatus (*f. c.*). The region of the direct cerebellar tract is also affected.

FIG. 2.—Section at the level of the sixth cervical segment, showing extensive degeneration in the posterior columns, leaving only a small layer of normal fibres in immediate contact with the grey matter of the posterior horns unaffected. There is marked affection of the lateral portion of the cord in the region occupied by the direct cerebellar and crossed pyramidal tracts, and marked degeneration on either side of the anterior median fissure.

FIG. 3.—Section through the mid-thoracic region, showing degeneration in the posterior columns, in the crossed pyramidal tracts, and on either side of the anterior median fissure.

FIG. 4.—Section through the upper lumbar region, showing considerable affection of the posterior columns, some involvement of the crossed pyramidal tracts, and a little degeneration in the region of the direct pyramidal tracts.

Fig. 3.

Fig. 4.

Fig. 1.

Fig. 2.

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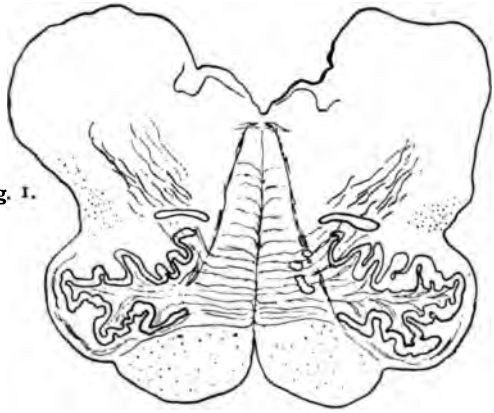
Fig. 1.



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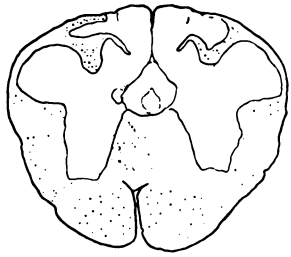

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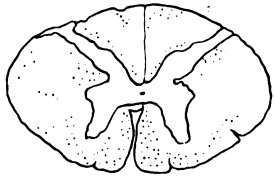
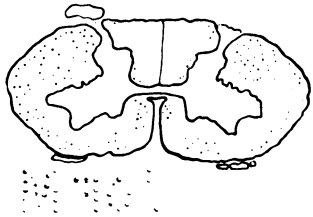


Fig. 4.



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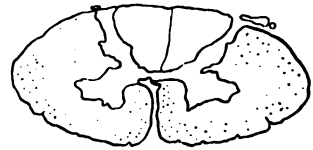

Fig. 6.

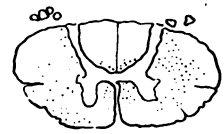

Fig. 7.

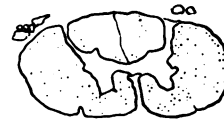

Fig. 8.

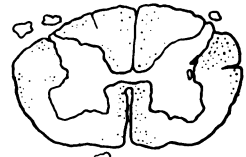

Fig. 9.

Fig. 10.

Fig. 11.

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DESCRIPTION OF PLATE II.

Illustrating Dr. Batten's case of Subacute Combined Degeneration of the Spinal Cord.

Series of sections through the medulla and spinal cord stained by the Marchi method. The sections have been drawn under an Edinger's projection apparatus, and the black dots represent the recently degenerated fibres.

FIG. 1.—Section through the medulla in the region of the olive, showing degeneration in both pyramidal and direct cerebellar tracts. A section at the same level stained by the Weigert-Pal method appears perfectly normal.

FIG. 2.—Section through the medulla at the level of the decussation of the pyramids, showing considerable degeneration in the pyramidal tracts, the direct cerebellar, and in the funiculus cuneatus; whereas the funiculus gracilis has no recently degenerate fibres. This section should be compared with the corresponding one stained by the Weigert-Pal method (Plate I, fig. 1).

FIGS. 3, 4, and 5.—Sections from the cervical region, showing the distribution of the degeneration. Fig. 4 corresponds to fig. 2 of Plate I.

FIGS. 6, 7, and 8.—Sections from the thoracic region, showing that there is considerable recent degeneration in those areas which appear normal by the Weigert-Pal method.

FIGS. 9, 10, and 11.—Sections through the lumbar and sacral region.

DESCRIPTION OF PLATE I.

Illustrating Dr. Batten's case of Subacute Combined Degeneration of the Spinal Cord.

Sections of the medulla, cervical, thoracic, and lumbar regions of the spinal cord stained by the Weigert-Pal method.

FIG. 1.—Section taken at the upper level of the pyramidal decussation, showing extensive degeneration in the funiculus gracilis (*f. g.*) and to a lesser degree in the funiculus cuneatus (*f. c.*). The region of the direct cerebellar tract is also affected.

FIG. 2.—Section at the level of the sixth cervical segment, showing extensive degeneration in the posterior columns, leaving only a small layer of normal fibres in immediate contact with the grey matter of the posterior horns unaffected. There is marked affection of the lateral portion of the cord in the region occupied by the direct cerebellar and crossed pyramidal tracts, and marked degeneration on either side of the anterior median fissure.

FIG. 3.—Section through the mid-thoracic region, showing degeneration in the posterior columns, in the crossed pyramidal tracts, and on either side of the anterior median fissure.

FIG. 4.—Section through the upper lumbar region, showing considerable affection of the posterior columns, some involvement of the crossed pyramidal tracts, and a little degeneration in the region of the direct pyramidal tracts.

Fig. 3.

Fig. 4.

Fig. 1.

Fig. 2.

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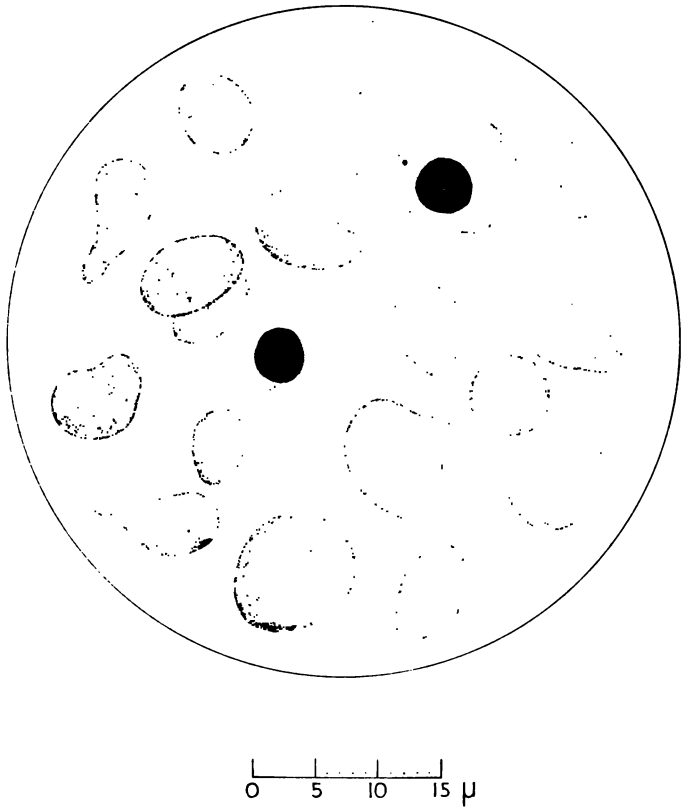
DESCRIPTION OF PLATE IV.

Illustrating Dr. Batten's case of Subacute Combined Degeneration of the Spinal Cord.

The drawing is made from a film preparation, stained with eosin and hæmatoxylin. It illustrates the great diversity both in shape and size of the red corpuscles. In the upper part of the drawing is a large red corpuscle containing a deeply stained nucleus. Below and to the left of this is a lymphocyte.

The film was stained and drawn by Dr. A. E. Phear.



A. E. Phear, M.D., del.

Bale & Danielsson, Ltd., Chromo-Lith.

lately been done in a paper published in 1898 by Dr. Russell, and again in a later paper published in 'Brain,' 1900. The view advanced there is that the condition is probably due to a toxic agent which is jointly responsible for the changes in the spinal cord and for the anæmia, and it is this view that would accord best with the facts in the present case.

Bibliography.—The bibliography of the subject will be found in 'Brain,' vol. xxiii, p. 108.

XIV.—*A case of Aortic Regurgitation probably due to Syphilis.* By CHARLES W. CHAPMAN, M.D. *Read January* 11, 1901.

C. L., married, æt. 40 years, came under my care at the National Hospital for Diseases of the Heart on January 5, 1899. She complained of fluttering of the heart, palpitation, giddiness, shortness of breath on exertion, and general feebleness.

Family history.—Both parents were living and about sixty-five years of age, and have always had good health. Five brothers and three sisters; one of the latter died in her confinement, the other seven are living and well.

Personal history.—She was very well up to her first confinement thirteen months after her marriage, twenty-two years ago. She has had no illness to her knowledge beyond weakness from her frequent miscarriages. She has had five children born at term, of whom two died of diphtheria at two years; the other three only lived a few weeks, and had snuffles and bronchitis.

The miscarriages (four in number) were from the eighth to the twelfth week. The patient noticed a distinct failure in her health about nine years since. Up to that time, beyond debility from her frequent pregnancies and miscarriages, she had not been actually ill; neither had she had any form of rheumatism.

Present condition.—Patient was very feeble, and had a worn appearance. On examination the action of the heart was found to be unequal in strength; the apex-beat was almost under the sixth rib, just outside the nipple line. The cardiac dulness was but little increased. A faint but distinct double murmur was heard in the aortic area; the other sounds were normal. The pulse was feeble, in other respects normal. Urine normal.

The case was put down as one of recent aortic regurgitation possibly due to syphilis. Mercury with iron was prescribed and persevered with for four months, when the gums became slightly affected. After a week's interval iodide of sodium and nux vomica were ordered. Eleven months after

the commencement of the treatment the heart-sounds were found to be free from any murmur, and they have remained so up to the present time.

On February 12 (thirteen months after commencement of treatment) a healthy full-term child was born, which the mother stated was by far the finest one she had had. The child has gone on well up to the present time.

Remarks.—Although I could not obtain any distinct history of syphilitic infection, I considered the evidence in that direction sufficiently strong to justify the line of treatment just detailed, especially as the more usual causes for cardiac or vascular disease were absent. As it appeared the patient had not had any antisyphilitic treatment, it was thought advisible to begin with a course of mercury.

Happily for the patient we have no post-mortem evidence of the correctness or otherwise of the diagnosis, but the clinical record is, I think, fairly conclusive. The points relied upon were:

1. The absence of rheumatism, gout, or chorea in the personal and family histories.
2. The patient had had no laborious work to do at any time.
3. The absence of renal disease.
4. The frequent abortions and the high mortality in the children born.

Sir William Broadbent,* when discussing the treatment of aortic stenosis, writes:— . . . "When, however, the valves are being damaged by a chronic inflammatory process due to syphilis, striking benefit may sometimes be obtained by the administration of large doses of iodide of potassium." Again, under the head of "Aortic Incompetence due to other Causes than Endocarditis," the same author states:—"Syphilis may be suspected as a cause of aortic incompetence which comes on in adult life or middle age, and is not traceable to rheumatic endocarditis or to gout or to high arterial tension. The regurgitation may rapidly become considerable, and as regards the character of the pulse, there will be no important deviations from the descriptions given. The principal difference will arise from the fact that the heart walls and cavities do not so readily take on compensatory changes; consequently the relation between the degree of dilatation and hypertrophy, which makes the latter approximately a

* *Heart Disease*, pp. 135—149.

criterion by which to estimate the former, no longer holds good. There may, therefore, be considerable regurgitation, with comparatively little enlargement of the heart."

Dr. Clifford Allbutt,* when writing on "Disease of the Aortic Area," observes thus:—"Syphilis is probably concerned in the causation of many cases of aortic disease, though, except when it exists in the form of a definite gumma, we have no certain test of the syphilitic process, whether in the living or dead body." Later on we read . . . "Yet, in deciding in a particular case that the aortic lesion is syphilitic, we are confined to the inferences which may be drawn from the story of the case, or from the associated changes elsewhere, which indications may, indeed, bring us to a moral certainty."

In some lectures by Professor Dieulafoy,† at L'Hotel Dieu de Paris, during 1896–7, the subject of syphilitic disease of the aorta is fully discussed. He considers this condition as most frequent and disastrous. He enumerates subacute and chronic aortitis, atheromatous and gummatous degenerations, and lesions of the aortic valve, and various aneurysmal dilatations of the aorta among the conditions engendered by syphilis.

He remarks how recently syphilitic affections of the aorta have been recognised by authorities. And he shows that the lesions have nothing special in their appearance, but that conclusive proofs of the syphilitic origin of them are in the efficacy of antisyphilitic treatment.

The author quotes a clinical case related by Leudet at the Blois Congress, in 1884, of obliterative syphilitic arteritis attacking successively the left and right frontal branches of the temporal arteries. He followed all the phases of the affection up to complete obliteration, and then observed the re-establishment of the circulation under antisyphilitic remedies.

Professor Dieulafoy relates a case of his own, of aneurysm of the radial artery, which completely yielded to specific treatment. He then describes the case of a man, aged forty-eight, where arterio-sclerosis was excluded, who had contracted syphilis eight years previously. This patient had aortic regurgitation and very severe angina pectoris; the pain was so distressing that, not only was he unable to follow

* *System of Medicine*, vol. v, p. 909.

† *Clinique médicale de L'Hotel Dieu de Paris*, 1896–7, p. 71, *et seq.*

his vocation, but the least movement brought on an attack of angina. Under treatment by subcutaneous injections of perchloride of mercury the angina completely subsided, so that he was able to resume his work. The aortic insufficiency, however, remained.

The author lays stress on the severity of the præ-cordial pain in cases of syphilitic aortitis.

In conclusion, I may be permitted to quote from Wilks and Moxon's *Pathological Anatomy*, published in 1875, the following passage:— . . . "Even at the present time it would save many valuable lives if syphilitic affections of the viscera were more readily and generally recognised."

XV.—*A case of Rheumatic Periostitis with Subcutaneous Nodules.* By Hugh Thursfield, M.D. *Read January* 11, 1901.

On November 1, 1900, a boy, æt. 9 years, was admitted to the Hospital for Sick Children for an eruption of subcutaneous nodules, and a swelling in the front of the right leg. His history was that two months before admission his leg swelled, and was red and painful, bad enough to confine him to bed for about a week. This attack left the bone of the right leg in the condition in which it still is: a uniform rounded, smooth swelling of the inner aspect of the tibia. This is now perhaps somewhat smaller than when first seen. Six weeks after this swelling made its appearance, two weeks before he came to the hospital, subcutaneous nodules appeared all over the limbs, head, and trunk; and as they showed no sign of disappearance his mother brought him for advice.

The boy is the second of three children, all of whom are alive. There have been no miscarriages, and there is nothing in the family history to suggest syphilitic infection. As to rheumatism, the only member of the family who has suffered from it is the boy's mother, who had acute rheumatism as a girl. Her heart is now quite natural. The child himself has had measles and whooping-cough. On admission there was found the periosteal swelling over the right tibia, and subcutaneous nodules on the scalp, forehead, elbows, tendons of the wrists, edges of the scapulæ, spines of the vertebræ, edges of the patellæ, and on the malleoli and tendons over the ankle-joint. They were at that time of the average size of a split pea, but the one in the middle of the forehead was much larger, causing a visible deformity.

Besides the nodules, which were of the type ordinarily associated with rheumatism, the only other evidence of that disease was an inability to extend the right knee-joint completely, due to pain at the insertions of the hamstring muscles, and a very moderate dilatation of the left ventricle of the heart. There was not then, nor has there been since, any cardiac murmur whatsoever. The boy at the end of a week in hospital had already begun to lose some of his nodules, though other fresh ones appeared, especially about the crest

of the ilia; but those on the scalp grew continually in size, reaching their maximum at the end of the third week. From this time they got smaller, and now cannot be detected.

The tendons of the hamstring muscles of the right leg had meanwhile become painless, and the leg fully extended; but on November 12 the tendons of the other leg suffered in the same way, and the joint became swollen, and for about a week contained fluid. There was some but not much pain, except on November 12, when the temperature in the evening reached 101·2°. Since the end of November there has been a gradual "regression," though some nodules still persist on the elbows and ankles, and once on December 6 he had a slight tonsillitis.

I do not think there can be any reasonable doubt that the manifestations in this case are all of rheumatic origin. The condition of the periosteal thickening in the right tibia of course suggests a syphilitic infection; but there is no evidence whatsoever of syphilis inherited or acquired in the boy's history, in the family history, or in the physical appearances. Further, rheumatic periostitis, though apparently rare, is not unknown. Dr. Garrod and Dr. J. A. Coutts, in November, 1889, presented to the Clinical Society an account of three cases of periostitis occurring in patients the subjects of rheumatic infection; in all three there were nodules present, and also severe cardiac lesions. The periostitis was situated in all three cases on the ulna. The authors referred to a few previous notices of the subject, and to these I have nothing to add.

As regards the nodules in the present case they were certainly more than usually numerous; but the chief point of interest appears to be the fact that with this diffuse eruption there is no grave cardiac mischief. It will be remembered that Dr. Cheadle regarded a profusion of rheumatic nodules as a sign of the worst possible omen, and always associated with severe endo- or pericarditis; and certainly it is rare to find a number of nodules without endocarditis. There are, however, cases recorded, notably one by Dr. Hadden in 1889, *Clin. Soc. Transactions*, vol. xxiii, under the title "Nodules without Rheumatism or Chorea." Such isolated instances do not, however, impair the general truth of Dr. Cheadle's statement, which certainly holds good in the majority of cases.

I have to thank Dr. Lee for permission to bring forward this interesting case; and Dr. Garrod for his reference to his and Dr. Coutts' paper.

XVI.—*A case of Pronounced Spleno-myelogenic Leucocythæmia subsequent to an attack of Malarial Fever.* By David Nabarro, M.D. (introduced by George Thin, M.D.). *Read February* 8, 1901.

MR. S. consulted Dr. Thin on account of enlarged spleen on the 28th March, 1900, at the recommendation of Dr. Perram, of Bedford.

Mr. S., who is æt. 23, went to Vancouver, British Columbia, in 1899, and considered himself to be then in good health. He stated that, after arriving in Vancouver (April 8th, 1899), he engaged in hunting, spending his time both on the mountains and in the valleys. During April and May, which were wet, cold months, he felt very tired. In July (1899) he was attacked by "chills." His account of the attack is that the rigors came on about midday, lasting an hour, then for an hour he felt hot in the head, with a final stage of perspiration. He also perspired during the night. He was confined to bed for four days. Not regaining his health he returned to England in October, 1899, after an absence of about eight months.

He gave no history of any febrile attacks since his return home. Mr. S. noticed that he was getting corpulent towards the end of August, 1899, and thought he was getting fat; he did not know that his spleen was enlarged until examined by Dr. Perram, when first seen by him on December 13th, 1899.

Dr. Thin wrote to Dr. Pybus for further information regarding the origin of the illness, and was informed by him that Mr. S. first consulted him on July 5th, 1899, and he was asked to see him again on July 11th. He then complained of feeling ill, had a quick pulse and furred tongue, and a temperature of 104° F. Under treatment—antipyretics and quinine—the temperature came down to 99·5° F., eventually becoming normal on the 19th. On the 26th of July he was considered cured.

During August and September he was subject to sensations of chillness, but had no rise of temperature. When he left for home on September 26th (having been in the country

for five and a half months), he seemed to Dr. Pybus to be suffering from some "pernicious toxæmia."

Mr. S. told Dr. Pybus that while at Manitoba, about April 2nd, he stayed two days in a low swampy place, and on July 20th to 27th he camped beside a small creek where there were swarms of mosquitoes. His brother, sister-in-law, and nephew camped here with him, and have all remained perfectly well. Dr. Pybus informs Dr. Thin that he saw three other cases of fever in a house two miles distant from that in which Mr. S. lived. These cases were similar in nature and were characterised by high temperature, 104° F. in one case, and 106° F. in a second. The attacks in these cases lasted ten, sixteen and twenty days respectively. They all made good recoveries. Dr. Pybus states that "these are the only cases of fever in the district during the ten years he has been in practice there, and were believed to be of a typho-malarial character."

On December 13th, 1899, when first seen by Dr. Perram, the enlargement of the abdomen was found to be due to splenic hypertrophy.

On March 28th, 1900, Dr. Thin found Mr. S. looking very pale, anæmic, and with a "very large spleen which descended to the groin on the left side and crossed nearly to the ilium on the right side." He made a very careful examination of the blood in order to discover whether any parasites of malaria were present, with entirely negative results, no parasites or pigmented leucocytes being seen. Finding that the proportion and characteristics of the white corpuscles offered a matter of interest, Dr. Thin asked me to undertake the examination of the blood in this connection, and by the kindness of Dr. Thin I saw Mr. S. on May 25th, 1900, when a complete blood examination was made, with the following result:

Colour very pale.

Hæmoglobin . .	36—40 per cent.	Colour index 0·6
Red corpuscles .	3,250,000 per c.mm.	
White corpuscles .	750,000 „	

Ratio being 1 white to every $4\frac{1}{3}$ red corpuscles.

The differential count of the white corpuscles gave the following percentages:

Small lymphocytes . .	6	per cent.
Large lymphocytes . .	4	„

Polymorpho-nuclear leucocytes	47	per cent.
Eosinophiles . . .	8	„
Myelocytes . . .	32·5	„
Basophiles . . .	2·5	„

The eosinophiles present were of the ordinary type, dwarf eosinophiles and eosinophilic myelocytes.

In all, 1030 leucocytes were counted, and while counting them 51 nucleated red corpuscles were seen; of these the majority were the ordinary normoblasts, with single nucleus staining deeply, while a few had two or three nuclei.

On this occasion (May 25) Mr. S. was rather pale, the lips and other mucous membranes distinctly anæmic. The spleen caused visible enlargement of the whole left side of the abdomen; it could be felt down at Poupart's ligament on the left side, and about one hand's breadth to the right of the umbilicus. A notch was palpable a little above the level of the umbilicus, and the patient was tender at this spot. The enlarged organ did not feel very hard. The liver could not be made out to be enlarged, nor were there any enlarged glands.

In September last Dr. Perram wrote to me that Mr. S. was better, had more colour, but that the spleen was as large as before. A differential count of the leucocytes was as follows:

Small lymphocytes . . .	1·5	per cent.
Large lymphocytes . . .	2	„
Polymorpho-nuclear leucocytes .	38	„
Eosinophiles	8·6	„
Myelocytes	46·5	„
Basophiles	0·8	„

Many transitional forms between the large lymphocytes and myelocytes, and between the polymorpho-nuclear leucocytes and myelocytes, were seen. There were fewer dwarf eosinophiles and small polymorpho-nuclear leucocytes than in the May count; the myelocytes had increased from 32·5 per cent. in May to 46·5 per cent. in September. The number of nucleated red corpuscles seen while counting the thousand leucocytes was only ten, whereas in May fifty-one were counted.

On November 16th Mr. S. came to see Dr. Thin again, to ask his advice about taking up some occupation, as he was then feeling so much better. I saw him on that occasion,

when his colour seemed rather better, and he was apparently less anæmic. The spleen was as large as when I saw him in May; there was no pain or tenderness over it, and no inconvenience from its large size, except a slight sensation of weight when patient was tired. There had been no hæmorrhages from any mucous surfaces, nor could any enlarged glands be discovered. Patient had had no "chills" of late, nor was there any temperature.

The results of the blood examination were as follows:

Hæmoglobin . .	40—42 per cent.	Colour index ·68
Red corpuscles .	2,966,700	
White corpuscles .	666,700	

Ratio of white to red corpuscles 1 to 4½.

Small lymphocytes . .	2·3 per cent.
Large lymphocytes . .	·5 „
Polymorpho-nuclear leucocytes .	38·4 „
Eosinophiles . . .	10·8 „
Myelocytes . . .	42 „

Basophiles very few, as were also the transitional forms between the large lymphocytes and myelocytes, while the transitional between the polymorpho-nuclear and the myelocytes were 5·7 of total. Nucleated red were 20 to 1000 white corpuscles counted. The condition of the blood at the different examinations is best seen from the following summary:

1900.	March.	May 25.	Sept. 8.	Nov. 16.
Red cells	...	3,250,000	...	2,966,700
White cells	...	750,000	...	666,700
Ratio white to red	...	1 to 4⅓	...	1 to 4½
Hæmoglobin per cent.	...	36—40	...	40—42
Colour index	...	0·6	...	0·68
Lymphocytes (small)	9%	6	1·5	2·3
„ (large)	10·6	4	2·0	0·5
Polymorpho-nuclear	41	47	38	38·4
Eosinophiles	10	8	8·6	10·8
Myelocytes	28	32·5	46·5	42
Basophile	1	2·5	·8	very few
Nucleated red (per 1000 white counted)	13	51	10	20

The films were fixed by immersion in alcohol and ether, and stained by eosin and hæmatoxylin, and by Ehrlich-Biondi triple stain.

Treatment.—As seen from the examinations of the blood, treatment has had little or no beneficial effect on the patient's condition. In March, Dr. Thin ordered Mr. S. to take Ferri

et Am. Citr., gr. v, with Liq. Arsenicalis ♏iij, twice a day; also every three weeks for a whole week, 2½ grains of quinine twice daily before meals. About October I suggested to Dr. Perram to try the effect of larger doses of arsenic, and he accordingly increased the daily dose to 21 minims of the liquor arsenicalis. This, however, soon produced toxic symptoms, so it was discontinued for a time. Since December Mr. S. has been taking Heminal tablets, gr. iij, three times a day, as well as Ferri et Amm. Citr., gr. viij, and Liq. Arsenicalis, ♏iij, c̄ Amm. Carb., gr. ij. Heminal is Parke Davis's preparation of hæmoglobin.

On February 2, 1901, Dr. Perram wrote that Mr. S. had improved considerably during the last month. His general health is much better, and his colour a little better, but there is no apparent alteration in the size of the spleen, nor can any enlarged glands be felt. He is taking red marrow in the form of tabloids or as "Marrol" (B. W. and Co.).

Remarks.—There are, I think, a few points of interest about this case worth noting. First, as regards the antecedent history, it appears that Mr. S. was quite well until he went to Vancouver, and although he was there for five months did not suffer from severe remittent or intermittent fever for a prolonged period. Although it has not been possible to demonstrate the malarial parasite in the blood or pigment in the leucocytes, the history of chills, followed by a hot stage, and later by perspiration, is almost, if not certainly, indicative of malarial infection. Sir William Gowers, in his article on "Leucocythæmia" in *Reynolds' System of Medicine,* found an antecedent history of malaria in 30 out of 150 cases, and a history of exposure to malarial conditions in 9 others. In a number of cases of this disease which have been in University College Hospital during recent years I have failed to find a malarial history in any case.

Secondly, the great size attained by the spleen before any attention was drawn to it, and even then it was accidentally discovered during a routine examination, and caused the patient little or no inconvenience, nor does it now except when the patient is tired.

Thirdly, the condition of the blood is rather interesting, for in March, 1900, the lymphocytes (large and small) formed nearly 20 per cent. or one fifth of the total number of leucocytes, and the myelocytes 28 per cent., or a little more than one fourth. In November (eight months later) the lymphocytes were only 3 per cent., whereas the myelocytes were

42 per cent., or nearly one half the total number of leucocytes. The polymorpho-nuclear leucocytes remain fairly constant throughout.

Lastly, although the total number of white cells in the blood is very large—nearly three quarters of a million per c.mm.—the patient is apparently in good general health, as well as anxious and able to follow an occupation.

XVII.—*A paper on Instantaneous Radiography.* By EDWARD W. H. SHENTON (introduced by J. H. BRYANT, M.D.). *Read February* 8, 1901.

THE apparatus employed in taking the skiagrams here shown is of a very simple nature. The coil is one capable of giving a ten-inch spark. The current that enters the primary of this coil is interrupted by a small steadily running electro-motor that, by means of a crank, dips a wire in and out of a vessel of mercury. The current is broken when the wire leaves the mercury. There is a suitable mechanism whereby the length of make and break may be adjusted, that is, make can be much longer than break or *vice versâ.* The maximum effect is got from a tube when the make is as long, in proportion to break, as it is possible to adjust it.

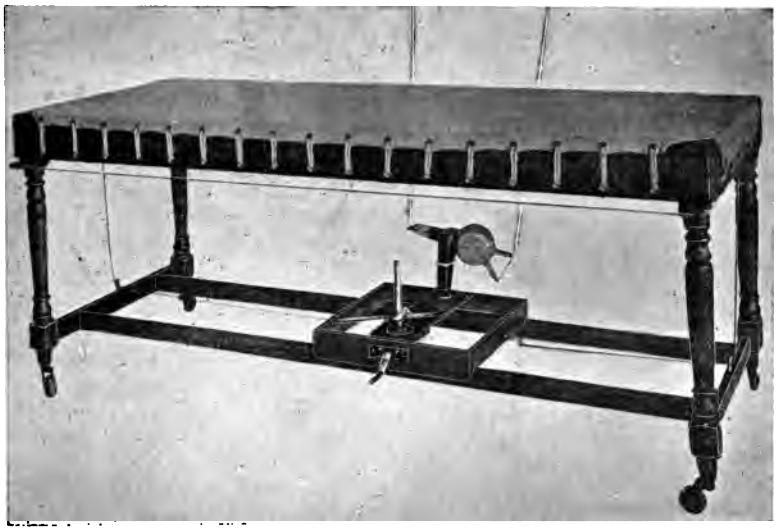
From the secondary winding of the coil come two chains which serve as leads to the tube. Chain is used in preference to wire, as it hangs parallel, while wire tends to get permanently bent, and so lessens the distance between the two terminals. The couch, which has been made from my design by Mr. Alfred Dean of Hatton Garden, consists essentially of a table, the top of which has been replaced by a tightly-stretched canvas, this material being practically X-ray transparent. The tube is placed in a holder beneath this table, and by suitable mechanism can be brought into any position by the operator while he is watching the change of effect upon the screen. This latter is placed upon the patient, who lies on the strained canvas. It is by these means possible to examine any part of a patient as he lies upon his back. The tube, which Mr. Dean has made from suggestions I have supplied, is very large in the bulb, and has two instead of, the more usual, three electrodes. Upon the careful exhaustion of this tube its efficiency depends.

The use of the word instantaneous is intended to convey the idea that exposure in radiography has been reduced to within reasonable limits. To be exact, exposure ranges from a fraction of a second to a maximum of thirty seconds.

This change has come about by the introduction of X-ray tubes of greater power. These are not only capable of giving

permanent photographic records in so short a space of time but, what is of far greater practical use, enable the deeper, portions of the body to be examined upon the screen, allow accurate localisation to be quickly performed, and fractured bones viewed during the process of setting. Since it has been possible to see upon the screen the deeper regions of the body, radiography has assumed more of the dignity of a branch of medical diagnosis. It is possible to examine a

FIG. 15.



patient now with the X-rays as quickly as with the stethoscope, and this is equivalent to saying that radiographic diagnosis is a practical as well as a useful art.

The following are notes of a few cases which will illustrate the advantages of this increased rapidity in the photography, but especially the greater capabilities of the fluorescent screen.

They are classed under three headings:—1. The detection of renal calculus. 2. Treatment of fracture. 3. The rapid localisation and extraction of foreign bodies.

The Detection of Calculus.

CASE 1.—Male patient, æt. 30, suffering with symptoms so

strongly suggesting tubercle of the kidney that he had abandoned his profession and booked a passage to Australia. While bacterial examination was being made of his urine, it was thought advisable to examine him with the Röntgen rays. He was placed upon the couch in the prone position, the tube being below the couch, and the screen upon the patient's back. A large dark mass was seen upon the screen which moved vertically about three inches. This was removed a few days later and proved to be a large branching calculus measuring two and a half inches in its longest diameter. A radiogram of this case showed no calculus. This was due to the movement which the stone made during the descent of the diaphragm in respiration. This movement would of course prevent a photographic record.

Case 2.—Female patient, æt. 35. This patient's symptoms were also strongly suggestive of tubercle of the kidney. Upon screen examination calculi could be seen in a semicircular arrangement. These were so obvious that they could be seen from the front as well as the back. The patient has refused operation up to the present. This photograph was exposed fifteen seconds.

Case 3.—Patient, æt. 14, complaining of pain in the left side and hæmaturia. A calculus was clearly seen upon the screen on the left side, and was removed a few days later. It was composed of oxalates and urates. This photograph was exposed twelve seconds.

Case 4.—Male patient, æt. 38, complaining of pain and passing large quantities of pus in his urine. He was supposed to be suffering from tubercle of the kidney. Upon the screen a large calculus was seen which upon removal proved to be composed of phosphates. This photograph was exposed twenty seconds.

Case 5.—Female patient, æt. 19. Symptoms were those of calculus. Two small calculi were seen upon the screen, and a photograph obtained in twelve seconds.

Case 6.—Patient, æt. 40, indefinite pain in the back. History of hæmaturia. Calculus seen upon the screen and recorded photographically in twenty seconds.

Case 7.—Female patient, æt. 40, had passed a small

facetted calculus. Examination showed what appeared to be upon the screen one fair-sized calculus, but which was in reality seven small stones aggregated so as to appear one. Exposure twenty seconds.

Case 8.—Male patient, æt. 45, with pronounced symptoms of renal calculus. The stone was seen very plainly upon the screen. The stomach, which was distended with gas, shows in the skiagram as a light area above the calculus. Exposure twenty seconds.

With regard to fractures and their treatment a great deal might be said of the improved results that are seen where a bright screen examination has been made during attempts to get apposition of the broken ends. One case only will be noticed here as an example of how screening may modify treatment.

Case 9.—A small girl fell upon the extended arm and sustained a fracture of the lower end of the humerus, the line of fracture being downwards and forwards. This injury is so common that 50 per cent. of all children's elbows examined in the Guy's Hospital Radiographic Department in the past year have shown this condition. Mr. Jonathan Hutchinson, jun., has pointed out how well the fragments come back into position when the arm is flexed, and how impossible it is to get good position with extension. Upon flexing this arm while screening, the lower fragment glided back into position, as is shown in the skiagram, and this position caused less pain than that of extension. This case has been since seen, and a little more flexion has produced a position which appears quite perfect.

Passing on to the localisation and extraction of foreign bodies, I will merely describe two typical everyday cases and the methods employed. The process for obtaining the exact locality of a foreign body which has been in use for the past two years in the Guy's Hospital Radiographic Department is performed without any special apparatus, and occupies but a few seconds.

Case 10.—Patient with a small pistol bullet in the right hand. The bullet had entered the hypothenar eminence, but could not be felt with a probe. The hand was placed palm

upwards upon the couch, the tube being underneath and the screen upon the hand. When the bones and bullet could be clearly seen, the tube was moved to and fro, and the moving image watched upon the screen. All parts of this picture did not move at the same rate, for those parts that were nearer the screen moved a shorter distance than those more remote. In this case the bullet, which was seen to apparently lie between the bases of the third and fourth metacarpal bones, moved at the same rate as these bones, or, what is the same thing, moved an equal distance.

It was inferred then that the bullet was upon the same plane as the bases of the third and fourth metacarpal bones. A probe was now held against the ulnar border of the hand, and was so arranged that the shadow of its tip and the shadow of the bullet moved at the same rate. It was then known that the probe was upon the same plane as the bullet, and therefore at an equal distance from the screen's surface. This distance was 4 centimetres. The hand was then turned palm upwards and the process repeated, and the bullet found to be situated 2 centimetres from the screen, or, what was the same thing, that it was 2 centimetres from the skin of the back of the hand. A mark was made immediately over the position of the bullet, eucain injected, and the bullet removed in a few minutes.

Case 11.—A needle in the knee. This needle was localised in the same way, save that each end of the needle was located. The process took one minute, and proved the needle to be in the joint. Mr. Golding-Bird subsequently removed the needle in the space of a few minutes.

Perhaps the most difficult case attempted was the localisation of a bullet lying against the neck of the femur in a muscular adult. The process occupied three minutes. As it is impossible to demonstrate this method with the rays to more than one or two people at a time, I have endeavoured by means of a transparent screen and limelight to give a rough idea of the principle.

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DESCRIPTION OF PLATE V.

Illustrating Mr. Shenton's paper on Instantaneous Radiography.

Showing a Renal Calculus in the Kidney of a man aged 45. **Exposure** 18 seconds.

DESCRIPTION OF PLATE VI.

Illustrating Mr. Shenton's paper on Instantaneous Radiography.

Showing a fracture of the lower end of the Humerus in a child, with an exposure of 2 seconds.

Fig. 1 exhibits the condition before treatment.

Fig. 2 shows the effect of flexion in replacing the fragment.

FIG. 1.

FIG. 2.

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DESCRIPTION OF PLATE VII.

Illustrating Mr. Shenton's paper on Instantaneous Radiography.

Showing the oblique fracture of the Tibia with an exposure of 2 seconds.

The fracture of the fibula was also oblique and situated in the upper third.

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XVIII.—*Some Personal Experiences of the Epidemic of Enteric Fever among the Troops in South Africa, in the Orange River Colony.* By H. H. Tooth, M.D., F.R.C.P. *Read March* 8, 1901.

Mr. President and Gentlemen.—It has been my privilege to be actively engaged in one of the most extensive and serious epidemics of enteric fever of modern times. Any value that the following recollections and reflections may possess must be attributed rather to their being the outcome of personal observation on the spot than to any particular qualification on the part of the observer other than may be claimed by a general hospital physician.

I must thank the Council of the Clinical Society most heartily for having done me the honour to ask me to open this discussion.

I wish to speak about myself as little as possible, but I cannot refrain from taking this opportunity of recording the extreme cordiality and courtesy with which I was received, wherever I was sent, by all ranks of the Royal Army Medical Corps, from the Surgeon-General downwards, and I cannot but believe that the intercourse, social and professional, that was established between the military and civil branches of our profession in the ups and downs of the great campaign, may help to lessen the distance which has insensibly grown between them, to the detriment of the service on the one hand and to our loss on the other.

I propose to limit my remarks this evening to a discussion of the enteric outbreak on the Orange River Colony side of the campaign, from the following points of view:—

(1) The origin of the epidemic.

(2) The dissemination of the disease.

(3) The general precautions taken to limit the spread of the disease.

(4) Preventive inoculation.

(5) Some remarks on treatment in hospital.

(1) *The Origin of the Epidemic.*

It is, as may be expected, very difficult to assign a definite date for the first appearance of this insidious disease. But it was in the Modder River camp that we must look for the

first cases, and I was given every facility by the colonel and officers of the Guards' Brigade Field Hospital, with whom I was staying in February, 1900, for obtaining information on this point; and though, unfortunately, I did not compare the facts so obtained with the experience of the other Field Hospitals, yet I think we may take the experience of the Guards as being probably typical of that of the whole force. From the admission and discharge book of the Guards' Hospital we find that enteric fever made its first appearance about December 23—four men of the same company, known to be friends, reported sick at about the same time, and therefore may be assumed to have been infected at somewhere about the same date.

Up to this time dysentery was the predominant feature of the daily record of admissions, but after this date enteric fever almost replaces it.

It must be remembered that the Guards arrived in South Africa in the latter part of November, 1899, and they proceeded after disembarkation straight up to Orange River, where they halted for two or three days.

The battle of Belmont was fought on November 23, Enslin on the 25th, Modder River on the 28th, and Magersfontein on December 11. The Guards were in all these engagements. The long wait of the troops at the Modder began on December 12.

Now, of course, the date of infection of the first cases of enteric fever above alluded to must be entirely a matter of speculation. But putting the incubation period at the outside at three weeks, we are forced to the conclusion that the enteric infection took place in the vicinity of Modder River. That the disease could have been imported from England or even from the Cape seems highly improbable.

Modder River cannot be said to be a town or even a village. It consists of the station, a small hotel, which was the headquarters of the Staff, a store, and a schoolhouse, all near the station.

There were several other small farmhouses, and two or three little hotels, mostly on the banks of the two rivers.

The Modder and the Riet rivers join to form one river, which retains the name of the Riet, and near this junction the railway crosses the Riet by a fine viaduct wrecked by the Boers. The two rivers are picturesque, with well-wooded banks, and serve in peace times as a pleasure resort for the people of Kimberley.

Kimberley is regularly visited by enteric fever, and it is therefore possible that the soil about Modder River Station may have become contaminated. Jacobsdal is on the Riet, about ten miles above the camp, and it was known that there were cases of enteric fever there. There is, therefore, every possibility that men might have been infected by drinking the water of the Riet. But the main source of water supply was the Modder, and there was no known contamination of it at this time in its course above.

As we shall see later, though it is impossible to acquit the water supply of having a share in the origin, yet in all probability other agencies played a still more important *rôle* in the spread of the disease.

It is important to bear in mind that this outbreak at Modder River was probably the direct antecedent cause of the great epidemic at Bloemfontein, and that the troops that entered Bloemfontein were deeply tainted with the disease.

The P.M.O., Col. Townsend, kindly allowed me to see the daily return of enteric fever cases from January 29 to February 6; and though I think they underrate the true state of the case, yet they give some idea of the rate at which the disease progressed in the early days. Up to January 29 97 cases had been reported and 17 deaths, or 17·6 per cent. On February 6 the total had reached 156, and 27 deaths—a mortality of ten per cent. These figures seem small, but they are fairly evenly distributed over the whole camp, indicating a widespread incidence. The general advance began about February 11, Kimberley being relieved on the 15th. Jacobsdal was occupied on February 16, and the next day Paardeberg was surrounded. Ten days were spent around Paardeberg, which surrendered on the 27th. Bloemfontein was occupied on March 13—about a month after the move from Modder River. The possibilities of infection on this long march, especially during the investment of Cronje's laager, must have been very considerable, but the fact of the widespread infection of the forces at Modder River must not be lost sight of, and there can be no doubt that men were falling out sick with the disease every day from the first day of the advance.

The Bloemfontein epidemic should be regarded as the direct continuation of that begun at Modder River.

(2) *The Agencies concerned in the Dissemination of the Disease.*

An army consists largely of individuals of the age most vulnerable to enteric infection. It would be impossible to find

a more suitable medium for the growth and dissemination of the bacilli than an army of 40,000 young men of ages between nineteen and twenty-five. Given, therefore, a preliminary case or two, and from quite small beginnings indefinitely large and serious results may follow. We will now discuss at some length the various agencies by means of which this scourge may be spread over a camp or communicated by man to man.

Water.—The water supply of the camp at Modder River station included the Modder, the Riet, and certain wells. The river water was charged with thick, yellow mud which was so fine that it seemed never to settle, and which rapidly clogged any filter. The authorities used various means for purifying the water, and even caused wells to be sunk. But all these efforts were rendered futile by the action of the soldiers themselves, who appeared to prefer the untreated river water. One never passed the drift without seeing the men drinking from the river and filling their bottles. If therefore there was any enteric contamination of the rivers above, the water supply must be held to have had a considerable share in the propagation of the disease. The soldiers were also bathing all day in the Modder, but below the point at which the water was drawn for drinking purposes. No doubt many men with the disease on them would be bathing at times and might thus infect their comrades. There was a well at the Station Hotel from which the headquarter staff was supplied. This water looked clear and sparkling, but the chemical examination was less favourable even than that of the Modder itself.

At Bloemfontein the question of water supply was a very pressing one, and on my arrival there I was asked to serve on a Board to inquire into the water supply and drainage of the camps.

It will be remembered that the waterworks at this time were in the hands of the enemy, so we had to rely entirely upon local sources of supply.

These consisted of wells and the spring from which the city takes its name.

The spring is situated to the south-west of and close to the town. It consists of a bricklined reservoir of about ten feet square covered in by a small red brick building, evidently an old engine house. This all lies in a dip in the ground, and is enclosed by a paling. The veldt on this side of the city drains into two large natural ditches or dongas, which receive in their course numbers of smaller ones. These two dongas

meet not far from the spring, to form one large watercourse which runs through the town. In ordinary circumstances this is dry, but in the rains it and its tributary dongas become rushing torrents, as we had ample opportunity of seeing for ourselves. On the banks of these dongas were several camps; for instance, not 400 yards away was a field hospital in which were 60 enteric fever cases, and provided with open-trench latrines; moreover, a donga is a convenient dumping ground for all sorts of refuse.

There was no evidence or suspicion that there was any communication between the spring and the donga, which were at least thirty yards apart, but the field hospital above alluded to was ordered to be removed. Another action of this Board was to cause the provision of bucket latrines to all hospitals and other camps; this was a very important and salutary measure. The water of the spring looked good, but by chemical analysis I found that it contained a suspiciously high percentage of absorbed oxygen and a trace of nitrites.

In addition to the spring, the city and forces had to rely on wells, of which there were several. I have personal knowledge, however, only of those near our own camp, and these were of the tube variety sunk through about 100 feet of underlying rock. These wells gave a good supply of excellent water, and from the manner of their construction were free from any possibility of surface contamination. There were two of them at first, and the engineers soon sank another. But these were not sufficient for the needs of the whole camp, and so the spring continued to be used as well.

The water supply from the Modder reservoir was reopened in May, when the epidemic was moderating, and therefore can scarcely be looked upon as a factor of influence in itself either way. However, I made several chemical examinations of this water and found it very unsatisfactory, containing a large percentage of absorbed oxygen. Thinking that it might be contaminated in its course through the mains, especially after a long period of stagnation, I procured water direct from the reservoir, and still found it far from satisfactory.

It is, however, quite probable that the contamination was of a harmless vegetable nature. All these chemical analyses are open to the objection that they give no hint as to the real nature of the organic material. They should be regarded as danger signals only; a personal examination of the source and course of transmission is of much more value, but unfortu-

nately often impossible. It is quite obvious that a harmless water may give a very unsatisfactory analysis, while an apparently pure sample may contain many bacilli.

The Results of Atmospheric Disturbances.—A visitor to the camp at Modder River would not have to wait long to see a sand-storm, and one would be quite enough to satisfy any curiosity he might have had. The veldt, generally speaking, is a plain of sand in so fine a state of division as to deserve the name of dust. After the rains there is a certain amount of loose vegetation which binds the sand, and rain and sun also form a hard surface which is not, under ordinary circumstances, much affected by the wind. But by the incessant movement of horses, men, and vehicles the surface became so ploughed up that one might walk a mile or more ankle deep in soft reddish-yellow dust. From time to time a gale would spring up, and in a short time the whole camp would, on the brightest day, be buried in the darkness of a London fog, and sand and dust cover everything and everybody. On still days, even, it was rare not to be visited by a number of small whirlwinds, called sand-devils, which would pass slowly along, sucking up quantities of sand and any light articles, such as pieces of paper, carrying them to an astonishing height into the air and depositing them broadcast. The result of all this was that sand entered largely into every article of food, for the cooking was performed in the open air. The air at that time was intensely dry, so that it was not too much to suppose that all sorts of fæcal and urinary contamination must have been disseminated with the sand and dust. The same state of things obtained at Bloemfontein but in a minor degree, because the atmospheric conditions were generally quieter at that time of the year.

As a factor, then, in the spread of enteric fever, we must by no means overlook sand-storms, which are everyday phenomena in many parts of the world where the British army may be sent, and it is quite a question whether they have not been more important factors in this war than the water itself. For that the water contained enteric bacilli is a matter of speculation, but that enteric stools and urine must have been deposited in the latrines and other places by men in the early stages of the disease there can be no reasonable doubt.

Flies.—As may be expected, the conditions in these large camps were particularly favourable to the growth and multiplication of flies, which soon become terrible pests. I was told by a resident at Bloemfontein that these insects were by

no means a serious plague in ordinary times, but that they came with the army. It would be more correct to say that the normal number of flies was increased owing to the large quantities of refuse—dead horses, etc.—upon which they could feed and multiply. They naturally infest persons who are ill, but seem to be peculiarly attracted to enteric patients, hanging in loathsome groups around their mouths and feeding vessels. They were all over our food, and the roofs of our tents were at times black with them. It is not unreasonable to look upon flies as a very possible agency in spreading the disease, not only abroad but at home. Most of the temperature charts that had hung over the patients' beds were plentifully spotted with the excreta of flies, and on my return home Dr. Schölberg was so kind as to make cultivations from them. But though he obtained cultures of many organisms he did not isolate the enteric bacillus, which circumstance, however, as he says, is not a matter for surprise considering the age of the material (three months, at least).

It is a well-known fact that with the first appearance of the frost enteric fever almost rapidly disappears. The winter in South Africa is very mild compared with what we are accustomed to in England. It is true that the nights are cold, sometimes to many degrees of frost. But the days are warm and bright; for instance, in my tent at midday the temperature was rarely below 70° F., and sometimes as high as 80° F.; in fact, about the temperature of an English summer day. It seems hardly credible that the almost sudden cessation of an epidemic can be due to the effect of cold upon the enteric bacilli only. But there can be no doubt in the mind of anybody who has been living on the open veldt, as we have for three or four months, that the flies are extremely sensitive to the change of temperature, and that the cold nights kill them off rapidly.

On consideration of these points it is surely justifiable to assign an important share to flies in the spreading of infection.

Personal Infection from Man to Man.—Even in the best ordered London hospital it is, unfortunately, not uncommon to see enteric fever pass from patient to nurse, and so to other patients. On consideration of the mode of life of the soldier on active service one is struck by the facilities it offers for infection. Personal cleanliness is often an impossibility even for officers, much less for privates. There is barely enough water for drinking, let alone for washing purposes.

The men slept fifteen in a tent when there were tents, and that presents considerable possibilities for infection. Far be it from me to say that the soldier is naturally dirty, but there can be no doubt that he has no idea of hygienic matters. This is shown forcibly by our experience at Bloemfontein. We had a large number of officers down with enteric fever, and as the nursing at that time was very heavy, we were glad to avail ourselves of the help of the personal servants of these officers. They had not much to do, of course, with the direct nursing of their masters, but nevertheless a large number of them contracted the disease while with us. Of course some of them may have been infected at the same time as their masters. On the other hand, of our own orderlies, nine only took the disease in the whole seven months, though they were in constant attendance on bad cases. This may be due to protective inoculation, but it is also probably owing to their being alive to the ordinary precautions necessary.

Bearing in mind the insidious mode of onset of enteric fever, it is obvious that at any one time there must have been in these large camps many men still on duty with the disease on them, and therefore capable of giving it to their comrades in the close intercourse of camp life.

In conclusion of this part of my subject I cannot help remarking that, far from its being a matter of surprise that the epidemic was so severe, it seems to me, who have lived through it, a matter of congratulation that it was not more so, considering how many unavoidable causes were at work.

The probable Effects of Diseases antecedent to Infection.—Here seems to be the place to discuss shortly certain points of more than academic interest, but for which clinical data are at present, so far as I know, lacking.

It is known, experimentally, that the virulence of the bacillus becomes much increased by the presence in the blood of the toxins of other organisms, even of such as are found normally in the alimentary canal, as *Bacillus coli* for instance.*

I quote from Dr. Horton-Smith's lecture, "Sanarelli showed . . . . that all that is necessary is to inject into a guinea-pig at first a dose not quite sufficient to be fatal. The animal recovers, but for some time afterwards attenuated typhoid bacilli may be found in the purulent focus at the seat of inoculation. If now in this condition a dose of toxines,

* Horton-Smith, *Goulstonian Lectures*, 1900. This question is fully discussed with many references.

either of *Bacillus coli* or of *Bacillus proteus vulgaris*, be injected into the peritoneum, the typhoid bacilli at once acquire fresh virulence, the organism is invaded afresh, and the animal dies from acute experimental typhoid fever. More important still, on two occasions he was able to determine such a relapse by injecting small quantities of the toxine into the stomach and so into the intestines, thus exactly reproducing the conditions occurring in the human subject."

Here we have an important principle enunciated from the laboratory, but which opens up a field of speculation perhaps not altogether profitless.

It is conceivable, nay, more than probable, that every man in these great camps, ourselves among the number, may have at times received into their alimentary canals enteric bacilli, which may have grown and multiplied, but without producing the characteristic disease. If this be so we may assume the existence of an immunity such as a previous attack might establish, or as it is hoped inoculation may imitate, or a natural immunity that we wonder at but cannot explain. It may be that such bacilli never get through into the circulation, but are passed away harmless. It is almost allowable to go a step farther in speculation, and to admit the possibility of the bacilli actually circulating in the blood without causing the disease. This is not so chimerical an idea as may appear at first sight, for it is now well known that persons who have had the disease may retain bacilli in their tissues for indefinite periods, of years even, without harm to themselves.

But suppose, in the light of laboratory experience, some other toxin to come on the scene, then the bacilli acquire a new virulence, and the disease may assert itself. Such a starting-point might be supplied by a simple attack of gastro-enteritis, with the multiplication of bacilli normally existing in small numbers in the intestines. I need scarcely enlarge upon the possibilities of such a trouble among soldiers with bad teeth and improper food.

The enteric epidemic has so overshadowed in the public mind all other medical ailments that we are apt to overlook the fact that dysentery, or its milder forms included under the head of diarrhœa, was quite as prevalent among the troops as enteric fever was. It would be difficult to find a man who had not had diarrhœa for some time longer or shorter during his stay in South Africa. We were struck by the fact that in many of our cases the enteric fever attack

appeared in the course of dysentery. I strongly suspect that the two diseases may exist in the same patient, and that such a conjunction may be of very unfavourable omen.

A widely distributed almost universal dysenteric diarrhœa, a daily exposure to infection by enteric bacilli—here are the elements necessary for a severe epidemic of enteric fever. Mark the importance of such a generalisation if true.

Diarrhœa and dysentery in themselves proved as a rule very amenable to comparatively simple treatment, but in the light of the foregoing remarks the slightest diarrhœa becomes invested with a new importance, and officers and men should be warned to apply for immediate treatment at a time when the simplest measures may have the greatest effect. This warning is sufficiently warranted by consideration of dysentery on its own merits, but it becomes doubly so if we look upon it as a co-worker for evil with enteric fever.

(3) *Precautions taken to prevent the Spread of Infection.*

At Modder River some of the regiments—the Guards for instance—made efforts in the direction of purifying the water. One tried boiling and clarifying with a small quantity of alum. Another used filters (Berkefeld), having as many as seven large ones going all day, but the water was so thick that the cylinders became clogged every ten minutes by a thick coat of slime. The Guards Hospital made use of Pasteur filters. After filtration the water was most palatable, but the objection to the boiling process is that in a tropical climate the water takes so long to cool, and when cooled forms a very flat beverage. At any rate the men preferred the river water in its natural state, and unless sentries were posted along the banks of the two rivers it is difficult to see how they could be prevented from drinking from them, even if it were positively known that the water was polluted.

Five or six wells were sunk along the bank of the Modder, but did not seem to come into general use.

The latrines were of the open-trench variety, carefully inspected daily, and with sentries over them to see that they were used properly. They were continually being filled in and fresh ones dug. The water supply at Bloemfontein has already been discussed, and the important measure of substituting the bucket for the open-trench latrines mentioned.

In our hospital and No. 3 Gen. Hosp. at the base it was

the custom to disinfect enteric and dysenteric stools with 1·250 mercuric perchloride solution, to remove them to a shed set apart for the purpose, and there to mix them with sawdust for burning.

At Bloemfontein the stools were buried after disinfection.

On the whole it is difficult to see what more could have been done. It is easy to make suggestions, but after all the carrying of them out will ever be subservient to the exigencies of warfare.

(4) *Preventive Inoculation.*

The conditions of life of an army on active service, even in camp, may well fill the soul of the sanitarian with despair. It is therefore with hope that we turn to the question of the possible artificial immunity by inoculation, which has naturally evoked so much interest in this war.

The figures that I have to give on this question are relatively small, but they possess the value of being the outcome of personal observation.

Portland Hospital Personnel.—The Portland Hospital went out with the usual personnel of a base hospital for 100 patients, as shown in the table.

PORTLAND HOSPITAL.

	No.	Inoculated.	Not Inoculated.	Enteric.	Died.
Medical Staff	5	4	1	0	0
Sisters	4	0	4	1	0
N.C.O.s, orderlies and servants	32	24	8	9	1
Total strength	41	28	13	10	1

Of the non-commissioned officers, orderlies, and servants, 24 were inoculated by us on the voyage out, and 4 of the medical staff at the same time. All of these showed the local symptoms at the time—that is, pain, stiffness, and local erythema; 17 also presented well-marked constitutional symptoms, general feeling of illness, fever, and headache.

Nine of the orderlies contracted enteric fever. It is significant that two of these had the disease very severely,

and one of them died; both of these had refused inoculation. Of the inoculated cases 5 had the disease lightly, and two rather severely. One of the sisters also had the disease rather badly, and she had not been inoculated.

Patients admitted to the Portland Hospital.—Including the members of our own personnel above mentioned, we had under treatment at the Portland Hospital 232 cases of enteric fever, most of which came under our care while the hospital was at Bloemfontein. These were cases under our personal care, and do not include among them convalescents, who were entered in the admission and discharge book as patients, but who really only occupied beds for a short time, until a train could take them to the base. If these latter were reckoned the percentages would of course be lower.

The following table shows the numbers and mortality among inoculated and non-inoculated (the sister is entered as an officer).

Enteric fever.		Recovered.		Died.	
		Inoculated.	Not Inoculated.	Inoculated.	Not Inoculated.
				Per cent.	Per cent.
Officers . . .	34	21	12	0 —	1 = 7·6
N.C.O.s & Men	198	29	141	4 = 12·1	24 = 14·6
Total . .	232	50	153	4 = 7·4	25 = 14

Of the total number 5 had been inoculated at home or on the voyage out, and of these 4 died, making a percentage of deaths among the inoculated of only 7·4 per cent. One hundred and seventy-eight had not been inoculated, and of these 25 died, a mortality of those not inoculated of 14 per cent. The general mortality in enteric fever with us was 29 deaths out of 232 cases, that is, 12·5 per cent.

If we take the experience among the officers alone, the evidence in favour of inoculation is still more striking.

The officers, as a body, have presented themselves for inoculation in much larger numbers relatively than the men. This idea is expressed in our figures, but of course they must be taken as suggestions only. Of 198 N.C.O.s and men, 33 only had been inoculated, that is, 16·6 per cent. But of 34

officers (including the sister) 21 had been inoculated, or 61·7 per cent., and only 1 died, and he had not been inoculated.

It is scarcely surprising, with these figures, that my colleagues and I feel favourably disposed towards inoculation.

I saw myself many bad cases among officers and men, not in our own hospital, and I am aware that several have died who had been inoculated, but this does not alter the strong general impression gained by clinical observation that the disease takes a milder and more benignant course in the inoculated than it does in the non-inoculated.

General Remarks on Inoculation.—A few remarks on the general question of inoculation may not be out of place here.

The material used is a sterilised pure culture of enteric bacilli in broth. The broth contains, therefore, dead bacilli and a toxin, which toxin, however, seems to be almost inseparable from the bodies of the dead bacilli, for a filtered broth has little toxic power. The supply we took out was given us by Professor Wright, and was prepared in his laboratory at Netley Hospital.

The dose is regulated by the quantity necessary to kill a guinea-pig of a certain body weight This is generally about 1 c.c. or ♏ 15–18. It is therefore a virulent poison. Injected into the body, more or less severe local and constitutional symptoms ensue.

The local symptoms consist of pain at the seat of puncture (most often the loin), erythema and lymphangitis spreading from the wound.

The constitutional symptoms may include rigors, fever, headache, a feeling of more or less severe illness, and often a tendency to faint, all lasting from twelve to twenty-four hours. These symptoms vary very much in different individuals: in some there is merely a transient indisposition and a pain in the side; in others there may be high fever, temperature 103° F. or more, and all the appearance and sensation of serious illness. After inoculation for an indefinite period the blood may give the Widal serum reaction; in my own case it was present three months after, well marked, and was faintly marked thirteen months after.

The inoculated person is now supposed to be in a state comparable to that of one who has lately had enteric fever, that is, he has had artificially thrown into his circulation the same toxin which is produced in enteric fever, and which possibly influences the activity and growth of the bacilli. He is also assumed to possess, as a result, a certain degree of

immunity as it is believed is acquired by the individual after an attack of the disease. The liability to infection by enteric fever is so variable that we are justified in supposing the existence of an equally varying degree of natural immunity in different persons. Moreover, the toxic saturation point, so to speak, is also a variable one, as is exemplified by the tendency to re-infection or relapse so often seen clinically. I am inclined to think that when the reaction to inoculation is slight, it is probably because the person possesses a certain degree of natural immunity, while on the other hand a severe reaction indicates proportionate vulnerability to infection. In the former case, if the inoculated person takes the disease, he has it lightly, but in the latter severely.

If this be true the toxin reaction possesses a real value as a means of forecast. But again, if this reaction is any measure of the degree of immunity possessed by the individual, it is obvious that, after a pronounced reaction, one or more re-inoculations should be insisted upon. For all that is claimed for the process is a temporary immunity, and that often partial only. An attack of enteric fever itself does not do much more, for second attacks are far from uncommon.

Inoculation has not yet had a fair trial. Medical officers have been too uncertain as to its value to insist upon one injection even, let alone re-inoculations. It is to be hoped that the figures of the whole army, yet to be published, will indicate with some certainty its value in the direction of lowering the mortality, as those quoted in this paper appear to do; this will justify the medical profession in taking a more decided attitude on the question than it has at present been able to take.

(5) *Remarks on Treatment.*

It may seem almost an impertinence on my part, in this presence, to make suggestions on treatment; but my doing so may serve as a protest against the attitude in the present day of some of the profession, as well as the public, in assuming that the care of enteric fever devolves upon the nurse rather than the physician. I have no hesitation in saying that, in a disease in which danger threatens from so many points, watchful supervision by the physician and prompt treatment of symptoms is not only productive of comfort to the patient, but also contributory in a high degree to a successful issue.

I am of opinion that there may be many worse places in

which to undertake the treatment of enteric fever than a comfortable tent in the open air. In fact, there can be no doubt that a tent is superior to a great many buildings, and this should be borne in mind, in view of newspaper criticisms on the failure, by the medical authorities, to secure all the available buildings in Bloemfontein.

Diet.—One of our earliest difficulties at Bloemfontein was the scarcity of fresh milk. It becomes necessary in such circumstances to fall back upon preserved or condensed milk. There are one or two forms of sterilised milk which are as good and palatable as the fresh article, but they are expensive. The condensed milks of numerous brands are excellent for ordinary use, but it is doubtful whether they possess much nourishing power in illness. The common sweetened form is most unsuitable; patients had, as a rule, an intense objection to it, and it caused undoubtedly in some cases troublesome vomiting.

The other forms of nourishment, *e. g.*, arrow-root, beef-tea, etc., we had in plenty. Our principle in dieting was to use all forms of nourishment liberally when, in our opinion, it could be safely done. Most of our patients were thin and ill-nourished before contracting the disease.

For myself I have many years held the opinion that food may be used much more boldly in enteric fever than is the usual practice. The safety of its employment depends solely on the digestive power of the stomach. If that power is normal, as in many cases it is, meat, for instance, is not likely to reach the ulcerated small intestine in a form more dangerous than a glass of milk is. For milk naturally becomes a mass of curds in the stomach before peptic digestion takes place, and if this latter process is incomplete the undigested curds may be a real source of danger. It seems to me that one should be guided in this matter by the state of the tongue, the evacuations, and the appetite of the patient. If the tongue is moist and fairly clean, if there is no diarrhœa, that is, if the evacuations are no more in number than, say, two a day, and fairly consistent, and if there be no tympanites, and the patient has a strong craving for food, it is probable that his digestion will be good, and that a liberal diet, including, for instance, finely-minced meat, and even thin bread and butter, will not only be safe, but beneficial. On the other hand, the dry brown tongue, frequent curdy stools, and especially tympanites, and a disinclination for food, indicate a condition of stomach that makes careful

dieting imperative. Frequent small feeds of milk, peptonised or diluted with barley-water, Benger's food, etc., must be used with care, and the stools frequently inspected as a guide to the state in which the food reaches the intestines.

But the mechanical effects of undigested food in the small intestines are not the only ones to be feared. Experiment seems to show that the growth of the enteric bacilli is favoured, and their virulence enhanced, by the presence in the intestines of other bacteria such as are encouraged by the presence of undigested food, and themselves tend to produce gastro-enteric trouble; so that a slight gastro-enteritis may have a grave effect on the course of the attack. This has been justly emphasised by Dr. Horton-Smith in his 'Goulstonian Lectures' (1900).

Alcohol we used most sparingly, and only in the severer cases in which we feared heart failure, not at all in the milder cases. The small rapid pulse we considered the best indication for its use; not high temperature. We rarely gave more than three or four ounces, and never more than six in the twenty-four hours. The long-enforced abstinence of the soldier from all forms of stimulant renders him rather susceptible to its influence as a drug.

Digitalis and strychnine were of the greatest value; in one or two cases gradually increasing doses of the official Liquor Strychniæ were injected hypodermically to the extent of thirty minims in the twenty-four hours.

Headache.—This was one of the first symptoms that called for treatment, and five to ten grains of phenacetine generally gave speedy relief, or, failing this, a somewhat larger dose of antipyrine. In not a few cases this caused, during the sleep that followed, a profuse perspiration, with perhaps a slight fall in the temperature. Beyond the discomfort of this no ill result followed.

In some cases, however, this sweating was so annoying that it became necessary to add $\frac{1}{100}$—$\frac{1}{50}$ grain of atropin, which usually stopped it.

Insomnia.—This was one of the most troublesome symptoms, and the cry for hypnotics was a very common one. As a rule all that was necessary was a small dose of phenacetine, which proved as useful for this purpose as it did for headache. To patients who were delirious and constantly attempting to get out of bed we did not hesitate to give hypodermic injections of morphine, and in considerable doses, such as $\frac{1}{4}$ grain, repeated several times if necessary.

Mouth and Tongue.—The care of the mouth and tongue may appear a minor detail. It is not so in the estimation of the patient, and in the severer cases the dry, black, and cracked tongue and lips are more than a discomfort; the pain of taking food may positively prejudice the nutrition of the patient. Local treatment may be all that is necessary—mechanical cleansing or the use of mouth washes; and of these we found one containing potassium chlorate, glycerine, and iron perchloride the most useful. A mixture containing potassium chlorate and sodium bicarbonate given three times a day had a remarkable effect in cleaning and moistening the mouth and tongue, so much so that its use became a matter of routine almost.

Diarrhœa.—Diarrhœa is, I believe, less a symptom than a complication, and as such requires treatment. In the minds of some it is regarded as an effort of nature to remove deleterious matter. I cannot accept this view. For in all epidemics a considerable if not large percentage of cases have no diarrhœa, and these are, as a rule, favourable cases, whilst among the worst cases diarrhœa is most common. This is shown by the fact that, of our 29 fatal cases, 21 had more or less severe diarrhœa. Diarrhœa may be due to general enteritis consequent on extension of inflammation from the ulcerated patches to the general mucous surface of the intestine, but it is probably more often caused by an ulceration of the large intestine. There is no doubt that such ulceration is often found in the colon post-mortem, very commonly in the region of the cæcum, and in two or three of our autopsies there was found extensive discrete ulceration of the large intestine even to the rectum.

The treatment of this diarrhœa was comparatively simple, and undoubtedly of great benefit to the patient. Bismuth carbonate or salicylate and chlorodyne proved the most useful drugs, supplemented if necessary by suppositories of a quarter of a grain of morphine. In obstinate cases enemas of 10 per cent. boracic acid solution often gave permanent relief—a further hint that the cause of this symptom was in large intestine.

Tympanites; always a dangerous condition, rarely gave us any trouble; it is best treated by boracic injection. A small injection of about four ounces of water, containing two grains of quinine and half an ounce of brandy, would often cause the patient to pass flatus with great relief.

Hæmorrhage occurred in ten cases, on the average at

about the eighteenth day. In some it was very severe, and was repeated two or three times, and was so profuse as to cause blanching. It was almost invariably accompanied by a sudden fall of temperature, and invariably by a rise in the pulse rate. In only one was it found after death, not having been suspected during life; with this exception it never proved fatal, and though four of these patients died ultimately, no blood was found in the alimentary tract.

Hæmorrhage always yielded to, or after, treatment, which consisted of a mixture containing oil of turpentine and tincture of hamamelis, of each half a drachm, and tincture of digitalis 3 minims. This was given every two hours, or alternately every hour with a drachm of hazeline. One of us used hazeline alone, a drachm every hour, with apparently equally good results. At the same time morphine suppositories were administered. Under this treatment the stools rapidly ceased to contain bright blood, and in about twelve hours were free from blood altogether.

Perforation occurred in five of our patients, averaging at about the thirty-fifth day. The earliest day of occurrence was the nineteenth, and the latest the fifty-first day of disease.

The prognosis of this terrible accident is gloomy in the extreme, whether viewed from a medical or surgical standpoint.

There is reason to believe that in a very small percentage of cases the resulting peritonitis may become localised, but this happy event cannot be foreseen, because it seems that the symptoms are rarely if ever proportional to the extent of the peritoneal inflammation. The symptoms of peritonitis in the course of enteric fever are not those of the same accident in a comparatively healthy person. The local pain, for instance, is rarely so severe, and may be almost absent; so also is the characteristic position of body. Abdominal movement may be present and ample. The absence of liver dulness as a sign is frequently wanting But a rise in the pulse and respiration rates, and a sudden fluctuation of temperature, are almost invariable, and of these the former is the most constant.

The diagnosis having been made the physician has two courses of treatment open to him, either equally unsatisfactory.

The medical treatment amounts to the securing of absolute rest to the intestines, in the hope of plastic localisation of the

inflammatory process; and this, of course, is best obtained by the free use of opium. If the issue must be fatal, this treatment is humane if nothing else.

The surgical treatment is as unsatisfactory as the medical, but nevertheless it is a question whether it is not the duty of the physician to recommend it. It offers a slender chance of recovery, and it is possible that as time goes on this chance may be improved. The history of abdominal surgery warrants this hope. If surgeons had allowed the high mortality in the early days of abdominal operations to deter them from operating repeatedly, that method of treatment would never have attained the position it now holds. It is difficult, however, to imagine a more unfavourable case for operation than a person suffering from enteric fever, at any date of the disease. The operation itself offers no peculiar difficulties, but in the two that were attempted by us we were glad to get the patient off the table alive. In neither case was the operation performed until ten to twelve hours after the first symptom, and it is of course possible that if it had been resorted to earlier the result might have been more favourable. The best one can say is that the patients both expressed themselves as feeling more comfortable and quite free from pain, but nevertheless they died four or five hours after.

DISCUSSION.

The PRESIDENT: I shall assume that the Society has accorded to Dr. Tooth its thanks by its applause for this most able and precise paper. The best compliment we can pay him is to discuss it very carefully.

I see Surgeon-General Jameson here, and I ask him if he will favour us by making some remarks.

Surgeon-General JAMESON: Mr. President and gentlemen, I need hardly say that this account by Dr. Tooth of his experience in South Africa is of very deep interest to me, because for many years we have had enteric fever staring us in the face in that part of the world. Year after year there is a severe outbreak in Ladysmith and in Pietermaritzburg, and when this war began the probability of enteric fever becoming prevalent in the Field Force seriously engaged our attention. We drew up and issued from our office instructions to medical officers as a guide to preserve the health of the men against this fell disease. After all this experience I do not think a single thing can be added to the instructions contained in that paper. It was certainly unfortunate that some years ago the sanitary officer was abolished from the field army. Consequently his duties fell upon a number of medical officers of divisions. Under the conditions which existed in Bloemfontein, where the whole of his energies were directed to finding accommodation for the sick, who were coming in in hundreds—one might almost say thousands—it was not very likely he could have very much time to look into sanitary matters. But I anticipated the difficulty and danger that would ensue; so I appointed on my own responsibility a gentleman—Captain Hughes—and equipped him with a chemical and bacteriological laboratory of a portable nature. His instructions were to proceed to South Africa and watch enteric fever. He had the advantage of being in communication with Professor Wright, of Netley, and hearing all his views. Professor Wright was prepared to assist him in every possible way. He embarked with Sir Redvers Buller. Sir Redvers placed him on his own personal staff in South Africa. He was with Sir Redvers at the battle of Colenso, and was there killed in action. Gentlemen, I think that was a national misfortune. There is no doubt that we have very great difficulties to encounter in a place like South Africa. And in this connection I would like to merely allude to the personal equation of Thomas Atkins. Thomas Atkins is a peculiar man, who does not believe in sanitation. He is a thirsty individual, and he will drink water, or anything else that comes in his way, without regard to conse-

quences. As an example of this, I may mention that at Fort George, in Calcutta, there were two sources of water supply; one was good and the other was bad. In order to indicate which was the good water and which was the bad, the bad water-tap was painted red, to indicate danger, and a notice was attached to the effect that the water from that tap was not under any circumstances to be drunk, but to be used for ablution purposes only. The other water was indicated as good water which could be safely used for drinking purposes. Tommy always preferred the bad water. It seemed to put him in memory of old times, I suppose. At all events there it was; whenever an opportunity presented itself he would take bad water, if he had any choice. This also has a practical bearing, because when I was in Egypt we used to have outbreaks of enteric fever in Alexandria, and it was always worst in one particular barrack. The water, which was derived from the Canal, we knew was bad, and therefore instructions were given that under all circumstances it was to be boiled. But still the enteric continued, and it was found that, although that water was boiled and passed through native filters for the purposes not only of filtration, but of aëration, the men went to the polluted source and had their drink of water from it. In this connection I may mention that in one barrack in Alexandria, in the most unhealthy part of the town, there was no enteric fever. The circumstances appeared to be exactly the same, but there was this difference—that the filtered water was handy for that barrack, while the other quality was at a distance, so that the men were practically compelled to use the boiled water. The whole circumstances of sanitation in connection with a field army are most difficult. I may mention that the Americans in their late war have had the same difficulties to overcome, and, if anything, in even a greater degree. They have found by their experience during the recent war with Spain that in standing camps, whatever the nature of the water may be—such as in the camp in Jacksonville, Florida, where the same water was supplied to the town as to the troops,—enteric fever was very prevalent amongst the troops in the camp, but the town was comparatively free. After a good deal of experience in that direction the system adopted in standing camps is to have a trough, and to put into that trough a considerable quantity of milk of lime, into which all the excreta of the camp is received. Then by means of a pneumatic suction engine it is received into a proper reservoir and carried away to a place of safety. My colleague, Major MacPherson, has some interesting facts on this subject, and he will be glad to communicate them to you. In the meantime I have to thank you very much on behalf of my colleagues and myself for the honour you have done us in inviting us to be present at the discussion this evening.

Prof. A. E. Wright (Netley): I wish, Mr. President, after expressing my appreciation of the honour you have done me

in asking me to take part in this discussion, to say at once that I do not propose to present the case for anti-typhoid inoculation to-night. Any adequate account of that treatment would require first an exposition of the method adopted in the preparation of the vaccine, and then a discussion of the principles of standardisation, and lastly, an account of the changes in the blood which are observed in animals and men after the inoculation of the vaccine. Not being able adequately to take up any of these points on this occasion, I propose, therefore, to confine my remarks to certain points which suggest themselves to me in connection with Dr. Tooth's opening paper.

I may begin by considering Dr. Tooth's observation that the object aimed at by the anti-typhoid inoculations is to render the blood of the inoculated person as like as possible to that of patients who have convalesced from attacks of enteric. This was, as a matter of fact, the object I set before myself in these inoculations. Taking it as established by general clinical experience that an attack of enteric confers a considerable—though by no means absolute—protection against reinfection, and taking it as practically certain that the protection which is acquired must be due to some alteration in the elements of the blood, it was obviously necessary to my object of assimilating the blood of susceptible persons as closely as possible to the blood of persons who had recovered from typhoid, to investigate as carefully as possible the blood of typhoid convalescents. To do this in a satisfactory manner new methods of investigation were needed. I turned my attention first to devising a method by which the agglutinative power of the blood could be quantitatively determined in a rapid and accurate manner. An appropriate method having been found, I set before myself as an object to attain in the inoculated an agglutination reaction which should be equal to that obtained in an average attack of typhoid fever. It was, however, manifest from the beginning that it was necessary to look beyond the achievement of a standard agglutination reaction in the inoculated patient.

Further methods for investigating the condition of the blood were therefore required. In particular there was required a method for testing in a quantitative manner the bactericidal power of small samples of blood such as can be drawn off from a patient. These technical difficulties having been overcome by the methods described by me in the *Lancet*, I set myself to apply these methods, with the help of Major Leisham, R.A.M.C., to the investigation of the blood of typhoid convalescents. Up to this date we have tested in all the bactericidal power of some 150 enteric convalescents in Netley Hospital. It might *à priori* have been expected that the bactericidal power of the blood of all these patients would have been found greater than that of the blood of normal healthy persons. We found, however, in conformity with the previous observations of Stern, conducted on a relatively small series of cases, and with less

precise methods, that while there is in many cases an increase of bactericidal power, there is in others a marked diminution.

If we take as an expression of the bactericidal power of the blood —and this is only one of the expressions which may be employed— the figure representing the extreme dilution of the patient's serum which is effectual in inhibiting the growth of typhoid bacilli when introduced in small quantities into nutrient gelatine, the bactericidal power of normal human blood would be measured by the fact that it will completely inhibit the growth of the typhoid bacillus in an extreme dilution of twenty, sometimes in an extreme dilution of forty. The bactericidal power of certain convalescents expressed in the same manner would be represented by the fact that a complete inhibition of the growth of the typhoid bacillus is obtained in a serum dilution of one hundred or even in greater dilution. In the case of other convalescents the bactericidal power is so reduced that the growth of the typhoid bacillus is not inhibited even in a two-fold dilution of their blood, this being the highest dilution up to the present employed. Unless we can bring ourselves to believe that such variations of the bactericidal power are quite without influence from the point of view of the growth of bacteria in the body, these facts afford much food for reflection. They would seem to confirm the opinion that patients may come out from an attack of typhoid fever with something to the good, in the form of an increased quantity of protective substances in their blood; such patients would obviously be patients who had elaborated protective substances in excess of what were required to secure a favourable issue. On the other hand, the observations referred to show clearly that among typhoid convalescents there are many cases to be found in which the patients who enter upon their convalescence not only with nothing to the good in the way of an increased amount of protective substances in his blood, but with an actual deficit of these substances. Patients in this condition would seem, so far as a very limited experience allows us to speak on the point, to be particularly prone to relapses. The variation in amount of bactericidal substances elaborated may obviously be due either to a difference in severity of infection, or to a difference in the constitutional capacity for elaborating their substances. I may instance in illustration of a probably defective constitutional power of elaborating protective substances the case of a surgeon on probation who recently came under observation at Netley. The history of this case was as follows:—He had suffered from a very severe attack of enteric fever, his health not being quite completely restored when he joined at Netley. With a view to protecting himself against possible future infections he underwent an anti-typhoid inoculation. Shortly afterwards, when stationed in Dublin, he again contracted enteric fever and two other inmates of the house developed the disease at the same time. As compared with these, his attack of enteric is reported to have been very mild. Believing

that the mildness of his attack was referable to the inoculation that he had undergone, I am informed that he reinoculated himself on his way out to South Africa. It has been reported to me through an indirect source, but I have not as yet been able to verify the fact, that the patient again contracted enteric fever in South Africa. We have here evidences of two, and possibly of three, attacks of enteric fever, not to speak of two inoculations, in the space of two years.

The application of facts such as these in connection with anti-typhoid inoculation is manifest to reflection. It would seem certain that there exists for every man a particular dose of the typhoid poison which is calculated to develop the maximum of response, and that the incorporation of a dose in excess of this is likely to be followed by a diminution instead of an increase in the bactericidal power. I have verified, among others upon my own person, the fact that a temporary decrease of bactericidal power may occur as the result of the inoculation of an excessive dose of anti-typhoid vaccine. Quite in harmony with this is the observation of Dr. Tooth, that the man who gives the severest reaction to typhoid fever does not always do the best. I am disposed to think that the patients referred to by Dr. Tooth, who show very severe reaction on the inoculation with the anti-typhoid vaccine, are patients who have received what is relatively to their powers of elaborating protective substances an excessive dose of the vaccine. The importance of the accurate adjusting of the dose of the vaccine to the capacity of the particular patient, or in the case of large bodies of men to the capacity of the average individual, will have become manifest, and this accurate standardisation is particularly necessary where a patient may be exposed to infection within a short time after the inoculation, for here it will obviously be important to avoid the production of a negative phase of increased susceptibility within the period immediately subsequent to inoculation. I shall return in a moment to the consideration of this question.

The next point I desire briefly to touch upon is the question of the results obtained by the anti-typhoid inoculations. Unfortunately there are up to the present very few statistical data available from which the results of these inoculations can be judged. Such statistics as have come to hand may, however, be briefly detailed

The first inoculations (apart from those undertaken for experimental purposes) were carried out by my then colleague, Major Semple, R.A.M.C., at Barming Asylum, during the Maidstone epidemic. Some eighty-four warders, attendants, and nurses were inoculated shortly after the enteric fever had broken out seriously in the asylum, and twelve of the staff had been attacked. One hundred and twenty warders, attendants, and nurses remained uninoculated. In spite of the fact that no uninoculated nurses

and warders were allowed to come in contact either with the enteric cases or with their effects, four of these contracted enteric fever. None of the inoculated, many of whom were in charge of the enteric cases, contracted the disease.

Encouraged by these results, and encouraged also by the fact that an officer in the Indian Medical Service, who had previously been three times inoculated, underwent at his own request an inoculation with living typhoid bacteria without suffering any evil effects, it was decided to apply the anti-typhoid inoculations very cautiously in the case of soldiers and officers in India or proceeding to India. The inoculations in question were carried out in particular by Major Firth, R.A.M.C. and Lieutenant Leonard Rogers, I.M.S.

Taking advantage of my visit to India as a member of the Indian Plague Commission, I succeeded in inoculating considerably over 3000 soldiers. This series of inoculations was carried out under circumstances of great difficulty. Much of the typhoid vaccine employed was prepared by me in the course of travelling from place to place; the standardisation had to be undertaken in the course of the actual railway journeys, the necessary guinea-pigs being carried with me in my railway compartment. Again, in default of appliances for ascertaining the continued sterility of the stock of vaccine after it had been opened for use, its continued sterility had to be provided for by repeated reheatings—a process which cannot be carried out without weakening the vaccine. I mention these details to show that I had no reason to be entirely satisfied with the quality of the vaccine. None the less, the official statistics which have come to hand with regard to the effect of the inoculations in these 3000 men—and with these are grouped another thousand inoculated by various medical officers, in particular by Colonel Fawcett, R.A.M.C., at Poona,—are not unencouraging. Of the 4500 inoculated, 0·9 per cent. contracted enteric, the percentage being 2·5 per cent. in the uninoculated, numbering some 25,000 in the same regiments and garrisons. In other words, in the case of the inoculated, one in a hundred had contracted the disease, whereas the proportion of men attacked in the uninoculated was one in forty.

I pass to consider the only series of official statistics which is at present available in connection with the anti-typhoid inoculations in South Africa. These refer to incidence of typhoid in the garrison of Ladysmith during the siege. They set forth that out of 1700 inoculated men, 2 per cent. contracted enteric fever, whereas of 10,000 uninoculated men, 14 per cent. contracted enteric fever; the incidence of enteric was thus seven times greater among the uninoculated as compared with the inoculated.

The above results apply to the officers and men conjointly. In contrast to these, the statistics relating to the officers separately set forth a larger percentage of cases and deaths among the inocu-

lated. The figures are as follows. Of 171 uninoculated officers 5 only are recorded to have succumbed to enteric, whereas of 44 inoculated officers 2 are recorded to have succumbed to enteric. These figures manifestly invited reinvestigation. When they were reinvestigated it was discovered that one of the officers returned as an inoculated, who had died from enteric, had been inoculated not with anti-typhoid vaccine, but with an anti-typhoid serum which had been obtained from a pharmaceutical agency. It was especially noted by this officer's relatives in connection with his inoculation that no symptoms had followed the injection. It was further discovered that the second officer who was set down as an inoculated officer who had died from enteric had been entered in this category through a clerical error. These rectifications place an entirely different aspect upon the statistics relating to the officers separately, inasmuch as no deaths from enteric appear to have occurred among inoculated officers. A similar rectification has to be made in connection with the uninoculated officers. It would appear that the number of deaths from enteric among these during the siege was 12, instead of 5, as set down in the statistics.

Passing to statistics available from the civilian hospitals which have accompanied the army in South Africa, the only statistics which have reached me, in addition to those which have been just set forth by Dr. Tooth, are those relating to the Scottish Hospital, which have been published* by Colonel Cayley, F.R.C.S., I.M.S., and those of the Irish Hospital communicated to me verbally by Dr. George Stoker. The statistics of the Scotch Hospital may be summarised as follows. The first section of the hospital staff consisted of sixty-one persons; fifty-seven of these were doubly inoculated on the voyage, two others being once inoculated, while the two who remained uninoculated had previously suffered from enteric fever. There was not a single case of enteric among the *personnel* of this first section, although they were very much exposed to infection, both in Kroonstadt and Bloemfontein, when enteric was at its worst.

The second section of the hospital staff consisted of eighty-two persons in all; nearly all these were inoculated, many, however, only once. There were in all in the second section of the hospital six cases of enteric. Three of these cases occurred among the small number of uninoculated; one of these in an uninoculated nurse, the only one (if we except two who had previously had enteric) of a batch of thirty-six nurses who had not been inoculated. The two further cases also occurred in uninoculated orderlies. Lastly, three cases occurred among inoculated orderlies; in each case the orderlies had been only once inoculated.

The statistics relating to the staff of the Irish Hospital contain little of special interest. Out of sixty-eight, sixty-four were inoculated, and one had previously had enteric. Among this *personnel*

* *British Medical Journal*, January 12, 1901.

there were, in spite of much exposure to infection, only two cases among the inoculated, with no death.

The last series of statistics which has reached me relates to the results obtained by the anti-typhoid inoculations in the case of the 15th Hussars at Meerut, who were inoculated shortly before proceeding to India in the autumn of 1899. The results of these inoculations appear from the following officially compiled statement.

FROM OCTOBER 22, 1899, TO OCTOBER 22, 1900.

	Strength.	Number inoculated in England.	Admissions to hospital for enteric fever.	Deaths from enteric fever.	Number not inoculated.	Admissions to hospital for enteric fever.	Deaths from enteric fever.
Officers .	22	19	0	0	3	0	0
N.C.O.s and men	481	317	2	1	164	11	6
Women .	36	24	0	0	12	0	0
Total .	539	360	2	1	179	11	6

It would thus appear that the incidence of enteric fever in the inoculated was represented by 0·55 per cent., and the mortality by 0·27 per cent., while the incidence in the uninoculated was 6·14 per cent., and the death rate 3·35 per cent.

With these I have detailed the whole series of statistics which are available up to date. I confess that I am not hopeful with regard to the prospect of obtaining any trustworthy general statistics relative to protective inoculation from the present campaign in South Africa. I would put it to you, not only that the war was not undertaken with a view to obtaining information as to the utility or otherwise of the anti-typhoid inoculations, but that the campaign has developed in a manner calculated to place extraordinary difficulties in the way of the collection of trustworthy statistics. The breaking up of the divisions into small detachments, the dispersal of these over an enormous tract of country, the perpetually recurring interruptions of the lines of communication, involving the cutting off of the outlying from the more central hospitals, the enormous pressure of work where typhoid has been epidemic, and the removals of the sick from the hospitals at the front to the hospitals at the base—all these would in any case have seriously interfered with the collection of statistics. When we add to this the absence of anything like a complete record of the number of inoculations undertaken, and the confusion which exists in the soldier's mind as to whether he underwent pre-

ventive inoculation against smallpox or preventive inoculation against typhoid—both of which operations were undertaken on the outgoing transports,—and lastly, when we take into consideration the difficulty of diagnosis in many cases of enteric, no very sanguine estimate can be formed as to the possibility of collecting trustworthy statistical materials on a large scale.

Passing lastly to the question as to how inoculation could be best made use of for combating enteric on a campaign, it will be manifest that in the absence of a trustworthy body of general statistics it is not possible to be dogmatic. Even at this stage, however, a few points seem to emerge from the examination of such statistical materials as are to hand, as well as from the scientific study of the effect of inoculation on the system.

1. It seems to be quite clear, both from the results of the blood-examinations, and from the results as regards protection recorded by Colonel Cayley, I.M.S., in connection with the Scottish Hospital, that the degree of protection afforded by two inoculations is much greater than that afforded by a single inoculation. It will be remembered that while the first section of the hospital staff, consisting almost entirely of doubly-inoculated persons, remained quite free from enteric, in spite of serious exposure to virulent infection, three cases of enteric occurred among the once-inoculated members of the second section of the hospital, which was much less exposed to infection. I need hardly again insist on the fact that, in the case of the employment of excessive doses, instead of benefit positive disadvantage might result from double inoculation.

2. It would seem from the list of cases and dates of attack furnished with the Ladysmith statistics, and also from the experience which I gained while inoculating in India, that the period in which the inoculated are least resistant to infection is the period immediately subsequent to inoculation. The proper policy of inoculation in the actual presence of an epidemic would thus seem to be to employ small doses of vaccine for the first inoculations, following these up for more complete and more durable protection by second inoculations with larger doses.

3. In the case of persons proceeding on service, and especially where inoculating only once with a relatively large dose, it would be expedient, where possible, to allow an interval of several weeks to elapse between inoculation and arrival at the theatre of war.

4. Lastly, re-inoculation should be resorted to after the interval of a year, more particularly in the case of men who have only been once inoculated.

It will be obvious that it was not practicable in the case of the present campaign in South Africa to comply with all these principles of action. In the first place, most of the troops were sent out to the theatre of war very soon after they joined the colours. The inoculations had therefore to be performed on the transport

in many cases only shortly before the men arrived at the theatre of operations. In the second place, in many of the transports only a very small number of men came forward for inoculation, and only a very small percentage of these underwent a double inoculation. It was manifestly impossible to expect it to be otherwise, inasmuch as the medical officers on board—in many cases these were civil surgeons—were of necessity incompletely informed on the subject of the inoculations.

I would point out in conclusion that the difficulties of inducing men to come forward *en masse* for inoculation, and especially for double inoculation, can only be overcome when the commanding officer and the medical officer are both alike zealous to effect everything that can be effected by the means of inoculation.

Sir Dyce Duckworth: My remarks, Mr. President and gentlemen, shall be few, and they shall relate to this question of inoculation, which I think is the real burning question in this very important discussion. I feel that not only this Society, but the whole profession, is indebted to my colleague, Dr. Tooth, for bringing forward this subject, because what has been said here to-night may possibly filter out beyond this auditory, and come to the knowledge of the British public, which reads everything and thinks it knows everything, and that some facts may come forward which may be of use.

With respect to this question of inoculation, sir, that is a subject I have taken considerable interest in, because from the outset I felt assured of its importance, and that we were likely not only to learn much, but to acquire by the practice of inoculation a useful means of preventing or rendering more amenable one of the most deadly diseases we know. Nothing has impressed me more, and I think I may say the whole profession, than the extreme of modesty with which Professor Wright has brought this matter to our notice, and the downright honesty with which he has supported his contentions by statistics. He has been most cautious and prudent, and has proceeded with his studies in this subject in a way that has inspired confidence in all of us, and will, I feel sure, rapidly bring out the great truths which lie at the bottom of this process. For my own part, as far as I have been able to read and see—and I have taken some interest in the matter—I have formed a high opinion of inoculation against enteric fever, and believe that it deserves large and extended trial, not only in naval and military centres, but among the general public. I think all nurses and persons who have to attend on patients with enteric fever should be submitted to inoculation, just as they are vaccinated against smallpox, and that people should be encouraged to come forward to undergo this process. I certainly think that statistics point strongly to the fact that a second inoculation is necessary; and that any data founded upon inoculations which have been made under rather doubtful circumstances, or where

inoculation has been practised only once, should in no way be allowed to vitiate the importance of the process. I think we should not consider that the practice has been fairly carried out unless two inoculations have been made. My belief is that if that process be carefully conducted according to the directions given by Professor Wright, a great amount of this disease may be avoided. The knowledge we possess at present distinctly encourages us, and shows it to be our duty to carry on and urge the adoption of inoculation far and wide.

Major MACPHERSON: Mr. President and gentlemen, I do not think I am called upon to say very much after what we have heard from Dr. Tooth, from the Director-General, and from Professor Wright, as well as from Sir Dyce Duckworth. But the matter brought forward has been one of extreme interest to me; and in listening to Dr. Tooth's paper one or two points occurred to me that it may be of interest to mention. One is that he has practically demonstrated from his experience in the Portland Hospital nearly every difficulty which it occurred to us would have to be faced in connection with the origin and prevention of enteric fever in the field. I am quite sure of the correctness of his statements with regard to the outbreak in Bloemfontein being due to the enteric fever, which commenced at Modder River camp; for the experience of all campaigns has been that enteric fever occurs in standing camps rather than with moving armies. No doubt this is due to the fact that an army is dumped down like a city on a place that is undrained, that is not regulated by sanitary laws, and that has none of the modern inventions for the elimination of enteric fever. If under such circumstances we could put in a drainage system, a water-carriage system, have sanitary inspectors, and apply sanitary laws, we might probably prevent enteric fever in standing camps. The Americans, who had a most extraordinary experience of enteric fever in standing camps—not in front of the enemy, but in their own States,—have come to the conclusion that, if you are going to put an army down in a camp, you must put down a water-carriage system and a system of drainage first. That is manifestly impossible in war, and they, recognising that fact, have, as the Director-General said, applied what is equivalent to a water-carriage system to their camps. They have established this system in their camps in Pennsylvania, and in a camp at San Francisco, where all the repatriated troops from the Philippines have been brought. Our own military attaché in Washington visited those camps, and has reported in very laudatory terms on their latrine system. The system simply amounts to this: the excreta are received into a trough, which is partially filled with a mixture of water and quicklime, or any other disinfectant. Proper seats are provided, and the latrines are similar to those in barracks at home, with this exception, that the excreta are not discharged into drains. Probably many of you know that the system of

latrines in barracks at home is that the troughs are partially filled with water, and are flushed out twice or three times a day. These American troughs are in exactly the same position as regards emptying, but, instead of emptying into the drain, an odourless excavator, such as one is accustomed to see in many continental cities, comes round two or three times daily, and the contents of the troughs are carried in water-tight carts to a distance and there deposited in the same way as the contents of drains might be deposited at a sewer outfall. We are endeavouring to get this system introduced into our standing camps, and possibly we may succeed. If so we shall advance one step towards the elimination of enteric fever from standing camps.

The question of the spread of enteric fever by sandstorms and flies was also anticipated by us, but it is extremely difficult to see how we can attack that form of dissemination of enteric fever. The instructions which we laid down before the campaign were that efforts should be made to destroy all refuse, etc., where flies breed and collect together; and officers were directed to pay special attention to this point. Whether the measures indicated could be carried out was a question which could only be dealt with locally. It may be of interest to note, as indicating the importance of this mode of spreading enteric, that, where the Americans used quicklime in their latrines, the cooks in the neighbouring kitchens found that the food became covered with quicklime from the flies, which came from the latrines to the kitchens.

Dr. Tooth said the open-trench system was substituted for the bucket system. We have had very great difficulty with regard to the suitability of trenches for the deposit of excreta. There is a general belief that if you deposit the excreta in shallow trenches you are doing much better than if you deposit them in deep trenches. But the experience in the army now is that shallow trenches, unless afterwards subjected to cultivation, are absolutely a source of danger. This has been fairly well brought out in connection with the prevalence of enteric fever at Quetta by the observations of Major Davies. He found that the water-supply was above suspicion, but that during a very severe epidemic the incidence of the disease was greatest in the huts which were nearest latrines. The further he got from the latrines the less enteric was encountered. In this case the excreta from the garrison were being deposited in shallow trenches, which were not cultivated; and they were to the windward of the barracks. The result of Major Davies' observations at Quetta is, that the excreta are now being buried in trenches ten and a half feet deep, and on a site to leeward. It is a question whether in campaigns we should not do the same thing, that is to say, if we cannot apply some form of water-carriage system similar to that in use in American camps. In fact the use of deep trenches is probably a better system in such cases than the bucket system—a system which the Americans say is just as

dangerous as the shallow trench system. Certainly the shallow trench and the bucket system give greater opportunities for dissemination of enteric by sandstorms and flies than the deep trench or water-carriage system.

There is another point which Dr. Tooth brought out, and which I think is of very great importance, but which has not been sufficiently noticed. I refer to the condition of the men's teeth. I have spoken to several men who had enteric in South Africa, and asked them how they thought they got the disease, or how they thought they got dysentery; and nearly every one said, "I think it is on account of my bad teeth." One had only to look at their teeth to see how really pathogenic the condition of their teeth might become as a nidus for germs. My own personal friends have asked me for rules to guide them in preventing enteric, and to all of them I urged, amongst the usual precautions, the importance of constantly using antiseptic tooth-powders or washes.

Professor Wright has given some statistics of inoculation, prefacing his remarks by saying that the subject was one that we were unprepared to enter into. The statistics which he quoted as to the result of the experience in India in 1899 were correctly given. But he did not give us the case-mortality. Now, the case-mortality of enteric amongst those inoculated is practically the same as amongst the uninoculated, as far as our experience goes at present. It is quite possible that these statistics may be vitiated by the fact that abortive attacks of enteric fever in the inoculated cases may resemble simple continued fever, and be returned as such; in which case there is something to account for the fact that the case-mortality of the inoculated patients, instead of being less, very closely resembles the case-mortality of the uninoculated. We cannot be too careful, however, in drawing comparisons from case-mortality. For example, Dr. Tooth gives this mortality as 12·5 per cent. in the Portland Hospital, dealing with small numbers. But evidence was brought before the Royal Commission to show the value of civil nursing and the general treatment in civil hospitals, on the strength of a low case-mortality. At the Civil Hospital, Bloemfontein, Dr. George Stoker stated that the case-mortality there was only 7·75 per cent. This statement has since been very much enlarged upon by Mr. Burdett Coutts in a letter to the *Times*, in which he spoke of it as "damning evidence" of the inferior way in which cases were nursed and looked after in military hospitals. That at any rate is practically what he said. Now there happened to be a military hospital in the same place, working during the same period—namely, No. 8 General Hospital—and the number of enteric cases in that hospital amounted to something like 2500 from April to the end of January last. The case-mortality amongst these cases amounted to 9·2 per cent., while the case-mortality of the Bloemfontein Civil Hospital for the same period amounted to 8·1 per cent. The Bloemfontein

Civil Hospital had comparatively few cases—258—and every one of the fever cases that were sent there for treatment was returned as enteric fever. There was, I understand, a strong opinion held by the doctor in charge of this hospital that all cases of simple continued fever were cases of enteric, and this probably accounts for the fact that all the fever cases were returned as enteric fever in his hospital. This would have an important influence in reducing case-mortality. Thus, if we add the cases of simple continued fever which were in No. 8 General Hospital to the cases of enteric, we get a case-mortality of 7·1 per cent. for the combined fevers, as compared with 8·1 per cent. in the Bloemfontein Civil Hospital. The figures thus come very near one another; and there is a similar experience in connection with the camps in America. The American Board of Investigation into the origin and spread of enteric fever included these undefined fevers in what they called "probable cases of enteric," and added them to the total number of admissions for enteric fever from these camps. They had then a case-mortality of from 7 to 8 per cent. for the combined fevers.

I think, therefore, that in making comparisons on case-mortality you have to take into consideration the mental attitude of the man who diagnoses towards that very moot question as to how far simple continued fever is enteric and how far it is not. Medical officers in the army see so much of these cases of continued fever alongside cases of enteric fever that they recognise very marked differences between them and enteric fever, and are not in the habit of returning them as enteric. This fact probably accounts for the comparatively high case-mortalities in some military hospitals; and may, as I have suggested, account for the comparatively high case-mortality of enteric fever amongst the men who have had anti-typhoid inoculations, in consequence of abortive enteric attacks amongst them resembling simple continued fever more than enteric, and being thus eliminated from the enteric statistics.

The PRESIDENT: I am sure the Society is to be congratulated on the very able discussion which Dr. Tooth's paper has elicited. We have several other names of speakers on our list, and it is therefore perfectly obvious that the discussion must be adjourned to a further meeting. The Secretaries will be much obliged if any who desire to participate in the discussion will send in their names.

Friday, March 22, 1901.

The PRESIDENT: We have now to resume the discussion on Dr. Tooth's paper on "Some Personal Experiences of the Epidemic of Enteric Fever amongst the Troops in the Orange River Colony." This very interesting paper was read at the last meeting, and in the interesting discussion which followed the question of the usefulness of inoculation against enteric fever was more especially touched upon. Many other questions arose out of the paper, and amongst

them was that as to the part which different kinds of flies played in the transference of the disease. I hope we may have some further light thrown on that point in the course of the discussion, and perhaps also in Dr. Tooth's answer to-night. A very interesting paper was published by Dr. Howard, of Washington, a little while ago, in which he recorded his experiences of the Spanish-American War of 1898, in which something like 80 per cent. of deaths were shown to be due to enteric fever, and he seems to have made a very elaborate investigation, not only on the effect of flies in carrying infection, but as to the varieties of flies which transferred infection especially. Dr. Howard observed something like seventy-seven species of fly which were concerned in this more or less, thirty-six of them actually breeding in excrement. He pointed out that the flies most concerned in the communication of the disease to the human subject were the common house variety, the vinegar fly, and the stable fly. I know nothing about the subject myself, but I thought if I mentioned it some of the members present might be able to give us some enlightenment upon it.

There was one other amongst the many interesting points raised which I should like to mention. That is in regard to the water-supply for the troops. As far as I recollect, Dr. Tooth did not make any special observation with regard to the conveyance of water in camps; but the subject is one of very great importance in reference to the possible origin of the disease. Through the kindness of Lord Dundonald, a commanding officer who has seen much service in Africa, and by the courtesy of the War Office, we have here to-night for inspection a model of a water-cart newly designed by Lord Dundonald. Its mechanism is fairly obvious, the object being to ensure perfect cleanliness. The receivers are, it will be observed, somewhat cigar-shaped, there being at the narrow end a screw plug provided with a tap for drawing off the water, and near the other end above a broad, funnel-shaped opening for filling, also provided with a screw plug. It will be noted—(1) that, the exit opening being at the end of the receiver and flush with its floor, (2) the receiver being so slung on the large wheel carriage that it can be easily tilted, and (3), the receiver being made of metal, it can be readily flushed and kept cleansed from stagnant depositions. Each receiver can be lifted from the carriage (which in this particular model supports three, arranged side by side) and placed on removable legs over a fire for boiling the water for making tea, etc., which can be drawn off at pleasure from the tap. I regret that Lord Dundonald has been unable to come himself to the meeting to-night to have made a few practical remarks on some details of camp sanitation from a military point of view, with especial regard to supply of wholesome drinking-water. The soldier must have drink, and it is for the medical military officers to devise means for its provision and conveyance in abundance and without contamination. I will now call upon Sir William Broadbent to renew the discussion.

Sir WILLIAM BROADBENT: Mr. President and gentlemen, the subject of the epidemic of enteric fever in South Africa is one of great interest, and I think it is well that it has been brought before this Society. It could not have been done in a better way than by the paper which Dr. Tooth has read to us. The number of cases of enteric fever actually dealt with is not very large, and therefore is not sufficient to serve as a basis for an estimation of the incidence of typhoid fever on the troops in a campaign, nor yet to serve as a means of estimating the severity of the epidemic as such. Our knowledge is not quite sufficient to establish the points in the causation of the disease which were considered by Dr. Tooth. These can only be done when the totals of the campaign are made up, and when the statistics are duly co-ordinated. But statistics always require interpretation, and the service rendered by Dr. Tooth, as a competent observer, will be especially valuable concerning the causation and prevention of the disease, and will serve as a guide to others who are engaged in working out these various questions. He himself thinks that the infection was originally contracted at the Modder River. There are not many of us who have the local knowledge to enable us to form an opinion on this point. Apparently these rivers are torrents at one period of the year and almost stagnant ditches at another, and it appears that at the height of the summer the Modder River and the Riet River were sluggishly flowing streams. I myself should not be prepared to exclude absolutely the possibility of the infection having been carried from home, notwithstanding the distance of time between setting sail, and landing. I think that people carry about bacilli sometimes for an indefinite period, without the destructive action of phagocytes and other agencies being actually efficient, and that then, if any very active exciting cause presents itself, there may be the occurrence of an attack of fever. Of course the interest of the occasion centres in Bloemfontein, where this epidemic was at its worst. Here a variety of causes may have been in operation. No doubt in many of the first cases the contagion was brought with the early arrivals, and later on there are the possibilities of the local water being impure. To these Dr. Tooth has added communication by dust and by flies, together with the direct communication from person to person, and of course the opportunities for such spread were greater here than in the well-ordered wards of a hospital. In a hospital this ought never, in my opinion, to take place, but it can scarcely be avoided under circumstances such as those which prevailed in Bloemfontein, owing to the great disadvantages which were there encountered. In India also the spread of typhoid fever has been attributed to dust, and I think much of the prevalence of the disease in some parts of India has been so explained. Communication by flies is a possibility, and it is to be hoped that some of these flies will be caught and sent home for examination; I do not think it was quite sufficient to send their

excreta. They might be collected wholesale, and I think it would be possible in that way to demonstrate the fact that they can act as carriers of the contagion. In this country, of course, we have always looked upon enteric fever as water-borne, and when in our investigation we fail to find that water has been the means of communication of the disease, direct or indirect—indirect in the way of watercress and oysters, and other molluscs—we have almost stopped short. It is possible that we may have, in the light of the facts brought to our knowledge in South Africa, to somewhat revise our ideas as to the means by which enteric fever may be spread. Other extremely interesting questions have been raised in the course of this debate. One of them is the antecedent predisposing conditions. These may be general debilitating influences. In this campaign we have men who were overworked and under-fed, but fortunately not suffering from the depression attending defeat. Another of these predisposing influences is gastro-intestinal catarrh, and digestive derangements generally. These must have been extremely prevalent. We are perfectly familiar with both these classes of causes in this country. We know that an attack of enteric fever may be determined by some depressing influence, or at any rate that some depressing influence or some gastro-intestinal derangement may render a person not previous susceptible to typhoid fever susceptible to it, and so bring about an attack. Then Dr. Tooth cited those specific predispositions which are brought about by dysentery and diarrhœa. I think there can be no hesitation in accepting his conclusion that such a specific predisposition was so generated. I think we see something of the same kind in this country in the case of influenza. I have come to the conclusion, from what I have seen in the course of practice, that influenza very frequently opens the door to enteric fever; that people get enteric fever after an attack of influenza—either immediately or a short time afterwards—who otherwise would have got rid of the enteric bacillus without developing an attack.

Another point brought before us in this paper was the intensification of the virulence of the enteric bacillus by other toxines. That, of course, has been illustrated experimentally. Not only does this happen, not only is there intensification of the virulence of the enteric bacillus, but the exudation resulting from the action of the enteric bacillus upon the intestines becomes itself a nidus for other forms of bacilli; and some of the symptoms of severe cases of enteric fever are not due to the enteric bacillus at all, but to toxines produced by other bacteria. In fact, we have here, as in tuberculosis—which you, sir, have called attention to—cases of "mixed infection," and sometimes the secondary infection becomes the more serious element in the disease.

Other questions of general interest arise as one considers this prevalence of enteric fever in South Africa. For instance, did Europeans originally take enteric fever to Africa, or did they

find it there? Has enteric fever arisen there from local conditions, independently of any importation by Europeans? A very interesting question, if it could be followed out, is the incidence of enteric fever on the native races. I do not know that there has been any evidence whatever on this subject. A sort of corollary to this question is, Is South African enteric fever exactly the same as the enteric in this country? Prof. Clifford Allbutt has told us repeatedly that there is no such thing as typhoid fever or as enteric fever. This is simply a forcible way of putting the fact that what we have to deal with clinically is not enteric fever, but individuals suffering from the invasion of the body by a particular bacillus. This being so—and no doubt it is true that we have to deal with an individual suffering from the effects of the typhoid bacillus—then we have had in this epidemic in Bloemfontein individuals under quite special conditions—over-worked and underfed, but, fortunately, with the one compensating advantage that there could be little or no excessive indulgence in alcohol. I think this fact that the soldiers could get little or no alcoholic drink has probably rendered the epidemic much less severe than it might otherwise have been. Then, also, the conditions affecting the bacillus outside the body are quite different in South Africa from those obtaining here. I suppose in these thinly populated districts, where some, at any rate, of this enteric fever occurred, there is no such thing as a sewer or sewer gases. The excrement is exposed to air and sun, which have a destructive influence upon bacilli of every kind. The outcome of these two special conditions surrounding individuals who suffered from the contagion ought to be some clinical peculiarities, and it will be extremely interesting, when we come to the close observation of the cases of enteric fever there observed, to note if there have been such peculiarities as we might be led to expect. I think, from the points which have come out in Dr. Tooth's paper, that this will prove to be the case. The question of the character of the bacillus is not without interest from various points of view. We know that in different epidemics in this country there has been evidently a great variation in the virulence of the poison. It is quite possible that these differences may be carried further, and, without there being any differences in the species of bacillus, there may be varieties, which may be of very great importance, affecting the protective influence of a previous attack of the fever, and possibly also seriously modifying the protective power of our artificial preventive inoculations. A nurse, whom I know very well, worked through the epidemic of typhoid fever at Maidstone without contracting the disease. She was preparing to go out to South Africa early in the campaign, when she got an attack of enteric fever, which she went through in St. Mary's Hospital. She successfully passed through the epidemic at Bloemfontein, but on going to Pretoria she got a severe attack of enteric fever which nearly cost her her life, The attack which she

had just before going out does not seem to have exercised a very long-continued preventive influence, and I think it possible that inoculations based upon attacks of the fever here, that is, where the preparations are made from cases in England, when we have to deal with the disease elsewhere, may not be very effective. If I were proposing to be inoculated as a protection against an epidemic of typhoid fever, I would rather that the vaccine were prepared from the products of the local epidemic itself. I think it is very likely that the protection *qua* that particular epidemic would be more efficient.

I cannot take up all the clinical points which were raised by Dr. Tooth, but there are two which I think have some importance. One is with regard to the symptoms of perforation. I quite agree with Dr. Tooth that the indications of perforation are not pain nor local tenderness, but extreme frequency of respiration and great frequency of pulse. It has occurred to me more than once to be called to cases of enteric fever with supposed acute pulmonary complications, when really what has occurred has been perforation of the bowel, attended with this extreme frequency of respiration. But Dr. Tooth says that in those cases which he has observed there was free abdominal respiratory movement. That has never occurred in my experience; the respiration has always been thoracic, and any abdominal movement observed has been due either to the traction on the abdominal walls by the thorax, or sometimes there has actually been a reversed action of the diaphragm. I have never seen true diaphragmatic respiration in a case of perforation, unless the patient has been at the time profoundly under the influence of opium. In all cases of peritonitis a large dose of opium may entirely change the character of this important symptom of peritoneal trouble.

The other point that I think it worth while to refer to concerns the treatment of hæmorrhage. The success which attended the treatment of that has been attributed to turpentine and to hamamelis: but I note, apparently as a secondary matter, that morphia suppositories were given. In my judgment the most important point in the treatment of hæmorrhage is to quiet the peristaltic movements of the bowel. When blood is being poured into the intestines it, as in the case of other intestinal contents, provokes peristalsis, and the peristalsis keeps up the bleeding and the bleeding keeps up the peristalsis. In that way the hæmorrhage persists. Whatever other treatment is adopted for this intestinal hæmorrhage, in my judgment the most important element is to quiet the peristaltic action by a dose of opium, either in the form of a suppository or given by the mouth.

These, sir, I think, are all the points that I need consider now. I will conclude simply by expressing my sense of the great value of this paper, and the service which this discussion will render. That service will be very considerable because Dr. Tooth's observations

will be a guide to others when we come to sum up the experiences of the whole campaign with regard to typhoid fever.

Sir WILLIAM CHURCH: Mr. President and gentlemen, I have been pressed to take part in this debate on the ground that I should afford it "local colour," whatever that term means. However, I do feel that I have this advantage over most, if not all, of those present—that I have seen the very spot upon which Dr. Tooth and the Portland Hospital conducted their labours, and I may say that of them I heard unanimously from everybody whom I met who had encountered them in South Africa nothing but the warmest thanks and praise. You will remember, sir, that the reference of the Royal Commission of which I had the honour to be one was that we were to go to South Africa to consider and report upon the care and treatment of the sick and wounded during the South African campaign. It was no part of our duty to investigate the causes of disease, or how disease might be avoided; it was only our duty to investigate the treatment and care of the sick and wounded in South Africa. Therefore I am not in a position to add much that is of value to the discussion to-night, and I am afraid not very much that is of interest. Still, there are a few points which perhaps I may interest the Society in giving my impressions of. Unfortunately I was not able to be present at the last meeting, but I have read with extreme interest the paper by Dr. Tooth and so much of the discussion as has been published. But it is quite clear, from what I have already heard from Sir William Broadbent, that points must have been discussed at that meeting which are not reported in any paper, and therefore if in any of the remarks I may make I repeat what you have already considered I trust you will excuse me. Our duty, as I have said, was entirely to report upon the care of the sick and wounded in South Africa, and we felt, considering the feeling that had been aroused in this country, that it was the duty of the Commission to carry that out with all possible speed, so far as it could be without neglecting any considerable points. It has been alleged that we hurried unduly over our work. Well, we all felt that the country was exceedingly anxious to know what was the state of affairs, and also what had been the state of matters which previously existed. That was the reason why the Commission worked with extreme haste—I do not think with undue haste—but I will say that they worked extremely hard. I am conscious that I am travelling a little out of the proper subject-matter of to-night's debate, but I would like the profession to know that on the very day the Commission arrived at the Cape they commenced their sittings. We arrived in the morning about 9.30, after which we of course had to make arrangements, and the Commission sat at 3.30 in the day. For the next thirty-five days there was only one single day—one Sunday—upon which we did not work, Sundays included. Not only did we work every day, but I might say the whole day, because while we

were travelling from one place to another there was a good deal of work to do. Therefore, so far as the medical aspects of the epidemic of typhoid fever were concerned, I can say very little from my own knowledge. The only point in the matter which really came directly before us was the sanitation of the hospitals themselves, which may really be looked upon as part of the treatment of the patients who were in them. But I trust that whilst I was in South Africa I took a fairly intelligent view of things, and I certainly kept my eyes open to see what I could, and my ears open to all I could hear, and I had many opportunities of extremely interesting conversation with both laymen and members of our profession. I would like to say that although I questioned almost everyone I met whom I thought capable of giving me satisfactory information on the subject of inoculation, including many nursing sisters and many professional men, as to the value of inoculation, the replies that I got were so contradictory that I really came away from South Africa without having formed any opinion whatever on the subject. Even before I went to South Africa I had formed a sufficient opinion of my own to act upon it, because during the course of the last two years the question has been not infrequently put to me, "Do you advise this young man, who is going out to India, or to South Africa as the case may be, to be inoculated for typhoid fever?" The answer I have always given represents my opinion, namely, I have taken some pains to find out the effects, and I can say that no one has been seriously injured by the process, though the subjects of inoculation are sometimes made exceedingly uncomfortable for a time, and I am inclined to think that it does afford a certain amount of protection from attack, and also renders it probable that an attack, if one comes on, will not be so severe as without inoculation. That, I think, seems to be a fairly general opinion. But when you press people a little further, and say, are you sure that the mortality of the cases you have seen has been less, you get unsatisfactory answers. You must remember that during the last year or so in South Africa there have been large numbers of persons who have been improperly and insufficiently inoculated, and therefore I fear that the result of the statistics which we may get from this war will be very unsatisfactory in the matter of inoculation.

Dr. Tooth stated, and I believe with perfect truth, that the typhoid fever which was the cause of the most distressing epidemic that occurred at Bloemfontein arose on the Modder. No doubt our troops were infected with typhoid fever at the Modder, so were the Boers; but no mention was made at the last meeting, according to the reports which I have read in the papers, of Paardeberg. Of course our troops marched from the Modder to Paardeberg and took typhoid fever with them, but it is quite clear to me that the great source of infection and poisoning was at Paardeberg. Not quite all, but almost the whole of our troops had to obtain water

from the river at a point lower down than that at which the Boer laager was situated, and I have spoken to several officers myself, who assured me that when they went to get water from the river —and there was no other water available—they were obliged, before dipping their kettle or bucket into the water, to push away a dead carcase. Of course that proceeding would not give one typhoid fever, but in addition to that there was all the filth of the laager, and not a few corpses of Boers who, it is not unlikely, had died from typhoid fever themselves. That, I cannot help thinking, was the great source of the original outbreak of typhoid fever.

I would like to break off here to consider for a moment—although Sir William Broadbent has mentioned it—something about the typhoid bacillus. When you think of it you will realise that we are extremely ignorant of its power and of its vitality, or even of its life-history outside the body. I think that laboratory experiments, so far as they show anything, indicate that in perfectly sterile sand—sand which contains no organic substances—typhoid bacilli will live but a very short time. In moist sand, but yet sand that is perfectly free from organic matter, they will live for some fourteen days. But laboratory experiments have also shown that in soil which contains organic matter, although it has been sterilised, typhoid bacilli will live and retain their virulence for at least 240 days. We really, I think, do not know how long typhoid germs may exist on the surface of the ground, or when they are only slightly below the surface, as when they are in dust. Neither, I think, are our bacteriologists quite decided as to whether the typhoid bacillus falls into the group of sporulating parasites. I think the general impression is that it is not a sporulating form of bacterium. But should that not be correct, should it be a sporulating form, we have no knowledge whatever of how long the spores may be able to retain their vitality, or under what circumstances they commence active life again. So I would just remind this Society that we are very ignorant with regard to the life-history of the typhoid bacillus, and I shall venture to assume to-night that the bacilli are capable of living for some length of time in dry media such as dust. That fact does assist one immensely in explaining the extent of the epidemic, and also other features, which I have scarcely time to enter into to-night, with regard to its spread and the catholicity of locality. The areas of visitation seem to be dotted about very largely, and I made a point of speaking to many of the practitioners who had resided for a long time in both the Orange Free State and the Transvaal. They told me it was a common occurrence to have a case or cases of typhoid fever at farms on the veldt which had no direct communication with any of the towns for weeks and weeks. The spread from one farm to another without such communication is capable of explanation on the assumption of aërial convection in dust. It is easy to imagine that dust can be scattered over almost any extent of

country. We were there in the cold months, and therefore I had no experience personally of the "sand devils" that Dr. Tooth spoke of, that is to say, whirlwinds which carried the filth out of the camps, but which had but a limited range. But I had an experience of the sand storms and dust storms, and one can imagine the typhoid bacilli being carried to almost any distance in those storms.

Passing from that, and following the epidemic from Paardeberg to Bloemfontein, at the latter place, so far as dust was concerned, there was the most favourable conditions for its propagation. A vast number of troops were there, and those persons who were examined by us, as well as those whom I spoke to privately, said that during the whole time, excepting when there were heavy rains, which were rare, there was a fog of dust during the movement of the troops.

I would like next to say a few words on the subject of flies, although I am equally ignorant with yourself, sir, on that subject. Fortunately for us, the winter weather killed nearly all the flies at Bloemfontein, but I might say that at Pretoria and Ladysmith we had sufficient experience of flies to form a slight opinion as to the scourge they must have been at Bloemfontein in hot weather. I spoke to many of the nursing sisters and many of the medical men on the subject of flies, for I have not the slightest doubt that flies constituted one of the ready means by which typhoid fever was spread at Bloemfontein. From all accounts, wherever the flies breed, and whatever may be their character, they do congregate around typhoid excreta when they have the chance, and certainly on typhoid patients. Many nurses told me that if one went into a tent or ward in which the patients were suffering from a variety of diseases, one could tell at once which were the typhoid patients by the way in which the flies clustered about their mouths and eyes while in bed. My own experience of flies, which is very slight, was that in Pretoria and Ladysmith it was impossible to keep them out of the food, even though they were comparatively few. One knows that at Bloemfontein everything eatable was covered in a moment with flies. These flies, of course, might have been in immediate contact with typhoid excreta on the veldt or anywhere else Therefore I think that we are not assuming anything that is improbable in thinking that flies play a very great part in the spread of an epidemic such as that at Bloemfontein. With regard to the sort of fly I did not learn very much, although I made inquiry. I inquired, as it appeared to me something might be done to prevent there being such a plague, because Bloemfontein before the army was there was not particularly infested by flies. Whether those who told me so were anxious that the place should not have a bad reputation I do not know, but they assured me that before the army went to Bloemfontein it was not at all remarkable for the number of its flies. Of course many flies live in decom-

posing animal and vegetable substances. but the fly which resembles our house-fly does not live on decomposing animal and vegetable bodies. I was told that a large number of this sort of fly are bred in horse-dung, and I think inquiries should be made on that point, because the amount of horse-dung about a camp is always very great, and more care might be used in collecting it together, and either covering it with lime or some other disinfectant, or at all events not rendering it available for flies. My opportunities of actually seeing the disease, except in regard to seeing patients lying in bed suffering from it, were not great, but I conversed with many men with regard to the character of the disease. Dr. Tooth put forward some extremely interesting suggestions in his paper with regard to different causes which might render the disease more virulent. Whatever may be thought of those suggestions as a whole, there is no doubt that the disease was very much more virulent in our men when at Bloemfontein than typhoid usually is in South Africa, That was the unanimous testimony of every practitioner out there. There is also no doubt that our men arrived at Bloemfontein in a very exhausted condition, and a great number of them with typhoid upon them. The men did not fall out of the ranks until they were absolutely obliged, and many continued in the ranks for a very considerable period after they were really suffering from typhoid fever. Again, even in those early days there were a number of cases of fever which were not recognised as typhoid fever, and even now one does not know whether they were typhoid cases or not. Still, it is probable that a large proportion of those men were subject to typhoid fever. Then, there were that large number of troops coming into Bloemfontein containing undoubted cases of typhoid fever, and a number of men who probably had it but did not know that they had, and did not fall out sick, and probably did not apply to hospital for treatment until some days after they reached Bloemfontein. Therefore, granting my assumption that dust has something to do with the spread of the disease, you have here exactly the conditions to cause the whole surrounding area to become contaminated. We think it probable that the bacilli voided by the urine are as virulent as those from the bowel, and you find, of course that patrols are going in every direction, and men having to fall out to relieve nature at other places than in camp. In that way one can easily understand how a large area around Bloemfontein would become contaminated. Grant that the bacilli are not all at once killed by sunlight and draught, and you then have here the very conditions in which the epidemic will spread rapidly, because of course the dust contaminates any open water in the neighbourhood, even if that water is not contaminated in other ways.

I should like to say a few words with regard to the water. I had not the opportunity of doing much walking there; but anyone

who has been in South Africa will know that even very little physical exertion, when there is sunshine and a hot wind blowing, will cause a most intolerable thirst; and to imagine that when soldiers come to a pool of water they should wait until the slow process of filtration has taken place is unreasonable. You would not do it yourself, sir. I had one or two fairly long walks, and the dryness of the mouth and the desire to drink is such that I think on one occasion I should have given way myself and drank at a pool of water, only I knew that at a little further distance back I had some drink in the carriage. In the same way with regard to boiling water, I suppose this on the table is an ingenious apparatus for boiling water on the march. But I do not know what fuel would be used. The question of fuel out there is one of extreme difficulty. I think you will be told that the men thought it their first duty to run about over the veldt and collect every patch of dry cow-dung they could find; and very proud was the man who could bring in more fuel than his neighbour. But all over the greater part of South Africa the absence of fuel adds very greatly to the difficulty of boiling water or cooking food. Of course I did not see these cases until after the virulence of the disease had passed; but what I was struck with was the mildness of the symptoms. In talking to the medical officers and walking the hospitals in September, what I was struck with was the large number of patients with undoubted typhoid fever; all except the worst were free from diarrhœa, although that freedom did not exist among the early cases to anything like the same extent as among the later ones. I think Dr. Tooth will bear me out when I say, as I only got the facts by questioning, that at the worst of times the number of cases of severe diarrhœa was not anything like so great as you would have expected from the general severity of the cases. That was a fortunate thing, because it made some of the undoubted wants and requirements and equipments of the hospital less distressing than otherwise would have been the case.

I should like to say a few words—if I shall not be tiring the Society—about what occurred to me with regard to prevention of the disease. Typhoid fever, ever since it was discriminated from typhus, has been known to be prevalent in armies, but its occurrence has not been quite so deadly as was that of typhus. But there has not been campaign, so far as I know, of any size in which typhoid fever has not played a very important part. The difficulties in the African army with regard to prevention are extreme, perhaps greater than in any other part of the world except India; if what we have assumed with regard to dust and flies be correct, that is one of the reasons and also the difficulty there is of obtaining good water. The Modder River has been mentioned. When I was there I crossed at the junction of the Riet and Modder rivers without so much as wetting my boots. That was at the end of the dry season. Of course there was water in the bed

of the river, and I had to pick my way from stone to stone, but there was very little water. I never got a satisfactory answer when I asked why more efforts were not made to use the Abyssinian wells, which were found so serviceable in the Abyssinian campaign. Very few attempts appeared to have been made in that direction. But artesian borings had been made, and some were very successful. Many of the rivers I saw consisted of a series of pools, and the water varied in appearance from that of yellow pea soup and green pea soup; but it was anything but inviting to drink, and had the disagreeable property of clogging every known filter in a very short time.

The prevention of typhoid fever depends, roughly, on sanitation. I regret very greatly that we had not the opportunity or time of seeing the condition of military camps; but our time was entirely occupied in seeing the hospitals and examining them. It is in the military camps that sanitation is most important for the prevention of disease; sanitation within the hospitals is almost sure to be good. Until this African campaign I believe that accompanying the army, almost always, or accompanying each division of the army, was a sanitary officer. I do not quite know the position he was in, but he was attached to the Headquarters Staff, and he had general supervision of the sanitary arrangements of the army or of the division. Several of the officers whom I spoke to agreed it was considered that the man in this position was of—comparatively speaking—little or no use, if, indeed, he was not a hindrance to the sanitation of armies on the march and in the field. That office was done away with, and the principal medical officers of the hospitals or on the lines of communication were then ordered to be the principal sanitary officers. This campaign has shown, I think, that it is very desirable that there should be sanitary officers appointed for all standing camps, or for all camps which are either within towns or in immediate connection with towns. It appeared to be the universal opinion of both officers of the Royal Army Medical Corps and of the Military that those were necessary, and Lord Roberts had appointed officers—or rather persons—to fill this office at Bloemfontein, at Johannesburg, at Pretoria, and possibly at other places; but there having been no regular provision for these officers of health—as I may call them—in these places, it was difficult to find a really competent person; they were competent in their knowledge, but for some reason or other they did not work well with the military. But it seemed to me that it was extremely desirable there always should be at any large standing camp a Medical Officer of Health, who should be responsible for the whole harmonious working together of the principal medical officers of the various divisions of the army, and also having charge of the intervening spaces of ground between the different regimental camps. The regimental camp officer is directly responsible for the condition of his cam to the Principal Medical Officer, whoever that may be; and

in another regiment the regimental officer is responsible for his camp, and so on. There seemed to be harmonious working of the whole thing, but we heard that there was neutral ground which it was nobody's duty to look after. Of the extreme value of a first-rate man of that sort I think no one who had an opportunity of seeing the work done, or of conversing with Dr. Turner, the Medical Officer of Health of Cape Colony, whom Lord Roberts telegraphed for and took to Pretoria, could have any doubt. There should be someone who should be in immediate communication with head-quarters, and should know from the Military when fresh troops are coming in, so that there should not be a chance of such accidents happening as did happen at Pretoria, where a regiment came in and encamped over the sewers of a hospital, the men actually using that water for washing.

I will make my other remarks very short. With regard to internal sanitation of the hospitals, of course when we saw them the arrangements were extremely good, but one could not help feeling that it would have been impossible for those arrangements to have been carried out with anything like the same thoroughness when the stress upon them was as great as it had been early in the year. I was extremely struck with the difference in the arrangements in the hospitals in the Natal portion from those in what was known as the central line. In the Natal hospitals—those from Durban to Pretoria—the scavenging of the camps was done by a special class of Indian coolies and sweepers, whereas on the central line—from Pretoria to Bloemfontein and De Aar—the scavenging was done by Kaffirs; that is to say, that part of the scavenging of the camps that the orderlies did not do. The difference, as it struck me, made, I think, a great deal of modification, both in the labours of the orderlies and in the value of their work. Whereas in the central line orderlies had to take the excreta themselves to the disinfecting shed, and also the vessels, to be disinfected, in the Durban to Pretoria line the orderly had merely to take the excreta and vessels to the door of the tent, where one of these coolies took it to the disinfecting shed and brought back the vessels disinfected, thus saving the orderlies an immense deal of walking and labour. I think it was found that the Kaffirs could not be trusted to do the work, whereas the coolies could. I think it highly desirable that there should be two classes of orderlies in connection with military hospitals. It seems a waste of power that well-instructed men should have to carry typhoid fæces 150 yards and bring the clean utensils back, and that there might be orderlies whose duties were entirely confined to hospital tents and to the feeding and nursing of the men, and others who should be the hewers of wood and drawers of water. Indeed, that would approximate somewhat to the arrangements at civil hospitals, where there are ward sisters, nurses, and scrubbers.

With regard to the prevalence of typhoid fever among the

natives, I made inquiries and found that, however they acquired it, the Kaffirs now suffer largely from typhoid. I spoke to Dr. George Turner on that subject, and he said that typhoid was the cause of many deaths among the Kaffirs, especially those returning from labour in Kimberley and other places.

Dr. ALEXANDER CROMBIE.—Mr. President and gentlemen, I am happy to be in a position to supply some of the information which was asked for by Sir William Broadbent—information of a clinical kind. I have had an opportunity during the last nine months of seeing a large number of officers who have returned from South Africa, who suffered from enteric fever there, and I will give a few statistics with regard to their cases. The first point is the frequency of second attacks among the cases in South Africa. Second attacks of typhoid fever are not unknown, but are not common. Berg gives only one case in 1624, that is to say, in ·06 per cent. But among the 150 officers whom I have seen there were nine instances of second attacks, *i. e.* 6 per cent They had previously suffered from enteric fever either in South Africa or in other parts of the world. In one case, that of a civil surgeon, typhoid fever was contracted in South Africa in May, 1900, and he then came home and convalesced; went out in September, and had a second attack in November. In another instance an officer had typhoid fever in India in November, 1899, and had it again in South Africa in August, 1900. That is the first point which I think remarkable in these case.

Next with regard to relapses. You all know that the occurrence of relapses in typhoid fever varies in frequency in different epidemics. Relapse occurs in from 4 per cent. of the cases to 16 per cent. In my 150 South African cases there were thirteen relapses, or 8 per cent., these figures occupying an average position. It is interesting with respect to inoculation to note how the process seems to affect the question of relapses after the disease. Of these 150 officers whom I have mentioned, seventy-seven, or 51 per cent., had been inoculated once; six, or 4 per cent., twice; nine, or 6 per cent., had a previous attack of enteric fever. The total was ninety-two or 61 per cent. of officers who had been more or less protected in these ways against typhoid fever. Six in these ninety-two cases, or 6·5 per cent., of those who had been protected against typhoid fever, had relapses; while of fifty-eight who had not been inoculated seven, or 12 per cent., suffered from relapses. This shows that relapses among uninoculated officers were twice as numerous as among the inoculated.

The next peculiarity in South Africa was the frequency of thrombosis. This complication occurs in less than 2 per cent. of cases in European experience. In South Africa it appeared in 25 per cent. of the cases; that is to say, one in every four had thrombosis more or less pronounced, generally in the left leg, sometimes in both, and once in the right leg only. As regards the effects of

inoculation, the protected suffered to the extent of 27 per cent., and the non-inoculated of 21 per cent. But there is no reasonable ground for thinking that inoculation would have any influence either on the severity or on the incidence of the sequelæ of enteric fever. The point which, in my mind, is the greatest peculiarity of the disease in South Africa, was the frequency of bowel irritation as a sequel of enteric fever. This has been of great persistence, having in some instances lasted for months. I have a case under treatment in a nursing home who had enteric fever in April of last year, and is still under treatment for this bowel irritation. In another case the enteric fever took place in May, and the patient is still suffering from similar bowel irritation. From the observations I have made clinically I have convinced myself that this bowel irritation is due to chronic colitis—a most troublesome and persistent disease. It is certainly a remarkable thing that whereas, usually, the condition of the bowel left after typhoid fever is one of constipation, here, in a third of the cases, there was the reverse condition, namely, muco-colitis.

There are four points in which enteric fever of South Africa appears to differ from what is usually regarded as the average sequence of events: first of all, the frequency of second attacks; (2) the frequency of thrombosis, which occurred in 25 per cent. of the cases; (3) the frequency of muco-colitis, occurring in one third of the cases to a greater or less extent; (4)—one which is a peculiarity I have not myself seen, but which I have heard of from medical officers who have served there—namely, the profuseness of the rash. These medical officers tell me that in that respect the cases differed from those usually seen in England and in India. Here one has to seek for the rash in typhoid fever very often, and if it is not seen on the abdomen one has to look for it on the back. But in South Africa in many cases the rash was distributed richly over the whole of the trunk as well as over the extremities.

As to the cause of this great divergence in the phenomena of South Africa typhoid fever as compared with those usual here, one has to fall back on the question, What is typhoid fever? We were told by Sir William Broadbent that it had been said that possibly there was no such thing as typhoid fever; but I think we have a sort of bedrock of fever upon which all the other symptoms arise. That bedrock of fever we have many opportunities of seeing. These are cases which run a mild course from start to finish, with practically no other symptoms—there is no rash, no diarrhœa, the spleen cannot be felt to be enlarged, there are no complications, and there are no sequelæ, but simply continued fever. This fever is of such frequency in large towns in India that it receives a distinctive name; the practitioners of Bombay are in the habit of calling it "Bombay fever;" and those in Calcutta "Calcutta fever," and so on. About eighteen months ago, before the British Medical Association, I spoke of it as "bastard typhoid." I think this is the

foundation of all typhoid fever, and that the rest of the phenomena in typhoid fever—the rash, the intestinal lesion, and so on—are the result, as Sir William Broadbent suggested, of mixed infection. Some very interesting experiments bearing on this point have been made by Rémy, and were reported in a recent number of the 'Annals' of the Pasteur Institute. That observer showed that when the typhoid bacillus and the *Bacillus coli communis* were cultivated together in the same medium, after the lapse of some considerable time they both lost their specific characters. The *Bacillus coli communis* loses its power of forming indol, and of causing fermentation in glucose; whereas the typhoid bacillus acquires the power of resisting its own antitoxin, that is to say, it is intensified in its power and vitality. Therefore I think it will probably be found that many of the phenomena of typhoid fever are not due to the *Bacillus typhosus* alone, but to co-operation with other bacilli which gain admission to the system at the same time from the intestinal canal. Sonarelli has found that when they are injected simultaneously, that is, the typhoid bacillus and the *Bacillus coli communis*, into the blood of an animal, the virulence of the typhoid bacillus is very much increased. I do not, however, think that the whole phenomena of enteric fever in South Africa can be due to increased virulence in the typhoid bacillus itself, because the mortality from typhoid fever in South Africa has been extremely low. When you look at this table which Dr. Tooth has prepared you see among the officers thirty-one cases and one death only, which gives a mortality of only 3·5 per cent. In England the death rate among the same class of men is about 10 per cent. Singularly the death rate among soldiers in South African hospitals was only 12 to 14 per cent. In England among soldiers it is 21 per cent., and in India 27 per cent. Therefore there is no evidence that the typhoid bacillus in South Africa was more virulent than usual, in fact rather less so. But the phenomena which I have mentioned are probably due to a peculiarity in its manifestations produced by the presence of other bacilli acting with it.

The PRESIDENT.—Our time is nearly at an end, and as we have a great deal of work yet before us this session, the question arises whether we should continue the discussion to-night a little longer—and I think that would enable us to finish it—or whether we should adjourn it. I must leave that matter in the hands of the Society. I myself think the probability is that we should get more interest out of the discussion if we tried to complete it to-night. Perhaps some member will make a motion on the subject. I think a continuation of half an hour would be sufficient for our purpose.

It was decided, by a show of hands, to continue the debate.

Captain WARD: I would hesitate at this late hour to contribute anything to the discussion, but I feel that a little practical experience may go a long way against the voluminous theoretical

ideas which have been advanced with regard to enteric fever and its prevention. What I have to say has reference to the disease as I saw it at Ladysmith during the siege. I came to the conclusion that very little was known about it. I was glad to hear that importance had been attached to the question of flies as a cause of the importation or the carrying of the enteric bacillus. I have special reasons for saying that, because the regiment to which I was attached—the 19th Hussars, from India—had been in India ten years, and were regarded as a seasoned regiment. They were the first regiment to develop enteric fever in Ladysmith, *i. e.* after we had been there six or seven weeks; and that regiment suffered more severely than any other. The water-supply of the whole garrison was practically from the same source, namely, the Klip River. The food was also the same, and yet I soon had cases which I suspected to be enteric. I consulted the medical officers in charge of other regiments, but at that time they had not noticed any cases amongst their soldiers. The enteric in my regiment still continued. The soldiers were not inoculated before they left India, because it was not possible to do it before embarking; the mobilisation was carried out too quickly, and it was not considered expedient to perform it on the voyage out, in view of the rough journey and our having a large number of horses on board. Under these circumstances it was not thought advisable to subject a number of men to inoculation, as it was possible that their services might at any time be urgently required. Enteric fever continued in that regiment in Ladysmith three weeks before any other regiment there developed it. I could not suspect the water-supply as the origin. The next regiment which had the fever was the one which was encamped next to us—the Liverpools. After them the next to take it were the Naval Brigade, who were our near neighbours; and from these three regiments the enteric spread all over Ladysmith. The Hussars, the Liverpools, and the Naval Brigade constituted the centre from which the cases radiated. This gave me much cause for anxiety and food for reflection. I investigated the matter, and found out that some years ago an enteric camp had existed at this spot; and I could only conclude that by some means or other (probably flies, because everything was a mass of flies) that the disease had been brought to the regiment by flies or such agency, and not by the water. I hoped that Dr. Tooth would have mentioned what was remarked upon by Colonel Crombie, the variations in the type of the disease as met with in South Africa, compared with that in England and India. I had not seen many cases in South Africa before I realised that the type was different to the normal. First, with regard to the onset of the disease. It did not, in any of the cases I saw, commence in the insidious way in which it is seen to commence in India or England. The patient was struck down suddenly, as we sometimes see him struck down by influenza. My own case was one in point. On the morning of

December 19th I was well, and had been down to the Field Hospital, assisted at operations, came back and had lunch, felt all right after lunch until well into the afternoon. Then I had a severe and intense headache, and when I took my temperature I found it was 104°. That night I was worse; next day worse again, and that was the beginning of my enteric fever. It also represented the history of the early stages of almost all the attacks which I saw in Ladysmith. The pain in the head was most intense, especially in the eyeballs; so much so that it was almost impossible to move the eyeballs without causing one intense discomfort. The onset was often accompanied by vomiting.

But with regard to the rash, Colonel Crombie has touched upon that, and that was another point upon which the South African cases have been very peculiar. The rash came out about the seventh or eighth day, sometimes earlier. Often it came out on the sixth or seventh or the eighth day, and its characteristic was the profuseness and wide distribution of the rash over the whole body, down the arms, and I have seen it in some cases on the palms of the hands and on the forehead. The severity of the rash did not appear to have any definite relation to the intensity of the attack. Sometimes with the most severe rash the case was of the most simple character and the patient got perfectly well. There was one case which I thought especially remarkable—that of an officer of the Naval Brigade, who had all the symptoms of early enteric. On the sixth day, when I was sent for, his temperature was 105·6°, and he was delirious. The following day I had a message to say that his temperature had fallen to normal. I suspected from that that he was going to have an attack of hæmorrhage. I found that he was a mass of rash; the whole body was universally dotted about with this characteristic dermatitis, and the temperature had fallen to normal. He had no hæmorrhage, but from that day he never had any rise of temperature; he convalesced and became well, the rash entirely disappearing.

With regard to the temperature, I always thought that the temperature in the South African typhoid was of a remittent type rather than of the regular course which one sees in cases of the disease in England. Frequently throughout the whole of the attack the temperature would be as low as 99° or 100° in the morning, and rise to 104° in the evening, this going on for the three or four days during which the patient was under observation. I do not think that is a type of temperature which one usually associates with enteric fever here.

The question of the number of second and third attacks of the disease has also been touched upon. My own servant had the most severe attack of enteric fever in Secunderabad, and it was not considered that he would live. He went to South Africa, and after he had nursed me during my illness he developed the disease himself and died on the tenth day.

With regard to precautions which can be taken to prevent enteric fever, all the elaborate things which have been told us about boiling and filtering of water, when you come to practical experience, are impossible. I feel sure it is not sufficiently realised at home how enormous the difficulties are in inducing soldiers to take means for the preservation of their health, even though you may provide amply for them. I always thought that the British soldier was as callous to enteric fever as he was to Mauser bullets and Boer shells. You may provide and filter water for him, but he will not take his fill of that—he will take it at the river. Therefore whatever methods for providing water and supplying it pure you may take, or whatever other precautions for preventing or limiting enteric fever, I do not think that when you come to bring practical experience to bear you will find any of them to carry water. There is one thing we do not do, but which I think we ought to do, namely, to cremate the cases which died of enteric. The mode of burial is very simple at present. The corpse is tied up in a blanket and let down into a few feet of earth. What the condition of the ground near the large hospital camps like Intombi or Bloemfontein are at the present moment one almost dreads to imagine; but I think it is possible that we are putting into the ground what will in future years be a grave source of danger to the population which might settle in the vicinity. I think something should be done in the way of putting some strong destructive agent into the grave with the object of destroying the bacilli, or, as I say, what would be better still, carrying out cremation.

As regards inoculation I shall certainly regard with scepticism any figures which come from South Africa in this point. The first reason is the enormous difficulties which exist in the compilation of statistics, and errors are bound to creep in. The statistics which we shall obtain will not go far enough; they will not show how many inoculations were done, they will not show the results of the inoculations, and they will not show the source of the serum supplied. In connection with this there is a very difficult and important question, namely, whether one person has acquired immunity from [illegible] the serum, which in one person [illegible] two, and in other [illegible] three [illegible] cases. I know of four cases in [illegible] when enteric fever [illegible] succumbed [illegible] Something must have happened [illegible] Whatever it is [illegible] Lady- [illegible] Whether another will [illegible] with cases [illegible] The point

I formed there—an opinion also supported by many medical officers whom I have spoken to on the subject—is that inoculation is distinctly favourable to cases of enteric fever; firstly, because the cases certainly have a much milder attack; secondly, because the mortality is less; and thirdly, that those inoculated cases which have not had enteric fever before have generally shown that the disease runs a very much milder course.

Dr. HILLIER: Mr. President and gentlemen, at this late hour I will not detain you at any length. The whole of this discussion has been of the greatest possible interest to me, inasmuch as I have spent the greater portion of my professional life in South Africa. I have been thirteen years in practice there, and during that time I saw a good deal of typhoid and other forms of fever, in towns, in mining camps, through a campaign, during hunting expeditions, and, in fact, under every conceivable condition of life in South Africa.

There are points in the case of typhoid in South Africa which no doubt might distinguish it to a certain extent from the typhoid epidemics of this country. Colonel Crombie has referred to a sort of unknown quantity, which he spoke of as the bedrock of this disease, as a form of fever which might have various complications due to mixed infection or other adventitious causes, and develop into typhoid or enteric or some other form of long-continued fever. I think if it be recognised that there is a tendency in these tropical and subtropical countries to get a form of fever which is mild in type, and distinctive and different from the milder forms of typhoid which you get in this country, we shall be nearer the truth. I know that Dr. Patrick Manson, in his work on 'Tropical Diseases,' distinctly states that there are a number of unclassified fevers in the tropics; and he says clearly that any conscientious diagnostician will recognise that there are cases which will puzzle description—cases of so-called simple continued fever which might last for two or three days or three or four weeks, and yet which will at no stage develop any of the characteristic symptoms of typhoid. I should like to ask Dr. Tooth whether in his opinion there is any distinction to be made between these milder cases and true typhoid. Colonel Crombie tells us that there are, in India, Bombay fever and Calcutta fever; and I may say that there are, in South Africa, Kimberley fever and camp fever, in addition to enteric of the recognised type.

I have been interested with regard to the observations that have been made as to the conveyance of the disease, and I have no hesitation in saying I believe that typhoid fever in South Africa on the whole is more usually conveyed by dust than by water. It was a regularly recurring disease in Kimberley. I was in practice in that town for seven years, and every autumn and late summer we invariably had a severe epidemic of typhoid, curiously climatic in its incidence; and yet the conditions of Kimberley are such as

almost to preclude the idea that in Kimberley it was conveyed by water. The water-supply of Kimberley was derived from the Vaal River, which was a large body of water practically running through an almost virgin country, and uncontaminated at least with typhoid germs. It was brought in eighteen miles to Kimberley. Nevertheless typhoid occurred, and with regularity. Now, the system of sanitation in vogue is the pail system, which in theory is an admirable system, but which in practice is abominable. There is always a carelessness in the carrying of it out, whether it be done by white men or by natives. That has always been my experience. There is also no doubt that it is a source of polluting the dust of the town, and probably by that means the disease is conveyed very considerably by dust storms. I recognise that polluted water is capable of conveying the disease, and does convey it in South Africa, as it would do anywhere else; but I think there is a disposition to attach too much importance to the filtering and boiling of water as a means of rendering it entirely innocuous. I do not believe the average water-supply is rendered innocuous by boiling or filtering, or both. I think the advice that Dr. Jameson gave to his patients in Kimberley was sound. If they asked what they should do with the water to make it safe the reply given was, "First filter it, then boil it, then throw it out of the window." What I mean by the danger which still exists in water which may have been both filtered and boiled is, that you may have various impurities only some of which are destroyed by boiling. For instance, for the sake of argument, if water contains sulphuric acid to a greater or less extent it is a noxious fluid, even after any amount of filtration; and so I believe that with a good deal of South African water it is a fluid which, if not capable of actually exciting typhoid, is quite capable of exciting that condition of gastro-intestinal catarrh which Dr. Tooth has suggested as a contributory cause of typhoid; what one may call a co-ordinate cause with the infection itself.

I will not say anything more about the clinical points which have been raised, but I have listened with a great deal of interest to the observations which have been made by Sir William Church. He made a recommendation which I am sure this Society will endorse, namely, that in future it would be desirable to have at all the great standing camps during a campaign an accomplished sanitary officer. One question which was raised at the last meeting, and replied to by Surgeon-General Jameson, was as to the desirability of having a bacteriologist at the front. Surgeon-General Jameson told us that a bacteriologist had been sent, and I think most of us are familiar with his name. We all, in common with the Department, deplore his loss, of which Surgeon-General Jameson truly said it was indeed a national loss. Every gallant life lost in South Africa is a national loss; but where a man is lost who cannot be replaced, it is more than an ordinary national loss, and

one cannot help asking whether the policy of sending merely one bacteriologist was on the whole a wise one. If it was a national loss to lose one, why should not half a dozen have been sent? In the same way I venture to think that this Society will also hold that, inasmuch as it was desirable and expedient for the Army Medical Department to do what the War Office had to do generally, to appeal to the nation, that is, to the civilian element in the nation, to co-operate in this great work in South Africa, which has cost the country one hundred millions, they might at least consider for the future the desirability, when supplementing the Army Medical Department, of taking in not only a large number of men from the rank and file of the profession, but also a certain number of senior leading physicians in the profession. It is perfectly clear that in every campaign of any magnitude disease has played, and does play, a very considerable part, even the greater part, in the mortality of an army in the field, greater even than the wounds of warfare. I venture to suggest, with all deference to the Department—and I make no charge against any individual or any department——

The PRESIDENT: I should be very glad if you would be good enough to keep to the subject immediately under discussion,

Dr. HILLIER: Certainly, sir; but I was discussing the policy which has been adopted, without assigning responsibility. Having lived for many years in South Africa I should like to thank Dr. Tooth for his very valuable contribution on the whole question of typhoid in that country. I can assure him his paper will be read with the greatest interest by the medical men there, and discussed in their medical societies.

Dr. HORTON SMITH: I think, sir, that it is absolutely clear from what we have heard this evening that the common soldier during a campaign cannot be expected to wait until his water is boiled or filtered before he drinks. It becomes, therefore, doubly important to prevent, if possible, the contamination of the existing water-supply.

In this connection, therefore, it may be asked whether all possible care was taken during this campaign to disinfect the typhoid excreta. So far as the fæces were concerned, it would appear from what Dr. Tooth has said that extreme care was directed to this point, and that, in general, disinfection was admirably carried out. Less attention, however, seems to have been paid to the urine, which, owing to the myriads of bacilli not uncommonly present in it, is really the more important excretion to guard against. Concerning the danger arising from the urine in many cases of typhoid fever, it may be of interest to state that the observations which I brought forward in 1897,* and again in 1900, demonstrating this fact, have received full confirmation from the independent observations of Richardson of Boston, Prof. Petracksky of Danzig, and lastly from the recent researches of Neufeld, working in Prof. Koch's laboratory,

* *Transactions of the Royal Medical and Chirurgical Society*, vol. lxxx, 1897.

in Berlin. It may be taken, therefore, I think, as completely proved that the fæces are by no means the only or, indeed, the most important way through which the disease is spread, but that in very many cases the urine is far more important and dangerous. The fact, however, hardly as yet seems appreciated by the profession at large.

But if this be so, it obviously becomes of the first importance not to confine our care only to the fæces, but to give our attention as well to the disinfection and sterilisation of the urine. How may this object be effected? Two methods are open to us. We may, of course, collect the urine after it is passed, add our antiseptics in sufficient quantity to it, and thus render it innocuous. The difficulties of carrying out such treatment during a campaign are obvious. Moreover, even at the best the method is unsatisfactory, since the soiling of sheets and linen with infected urine cannot thus be avoided. We are driven, therefore, to our second method, namely, the sterilisation of the urine in the bladder, so that when it leaves the body it is free from typhoid organisms. I desire to submit this evening that we do possess a drug which is capable of effecting this, namely, Hexamathylen-tetranin, more commonly known as urotropine. If this be given to a patient in doses of ten grains three times a day (as a mere preventive probably smaller doses are sufficient) typhoid bacilluria, if present, rapidly disappears, and does not recur so long as the administration of the drug is continued. This has been demonstrated in many cases by Richardson and by myself, and only two months ago has been fully confirmed in Professor Koch's Institute at Berlin by Neufeld. In this drug, therefore, we possess a means of preventing the growth of the typhoid bacillus in the bladder, and thus rendering the urine innocuous. Consequently, if it could be given throughout the disease, *until convalesence is well established*, to all patients, we might reasonably hope that the spread of the disease during a campaign would be considerably diminished, and that personal infection from man to man—by no means uncommon, as Dr. Tooth has shown—would become much more rare.

It may be justly asked whether there are any drawbacks to prescribing the drug in this manner. Speaking for myself I may say that I have given it in this way in several cases of typhoid fever, and have also taken it myself in these doses for a period of six weeks, but in no case has any harm been produced, nor has albuminuria ever occurred. The utmost that I can say against the drug is that if the urine happens to become concentrated, some pain and frequency of micturition may be experienced—a condition, however, generally temporary and requiring no treatment; but in any case easily obviated by omitting the drug for a day or two, or diminishing the dose. I should, however, add that two cases have been privately reported to me in which hæmaturia is stated to have occurred during the administration of the drug (and *possibly* as the result of

it) so that it is well to examine the urine carefully from time to time whilst the drug is being administered. Such cases, however, are clearly exceptional, and in future campaigns, and indeed, also at home, especially where the poor have to be treated in their own houses, and where, consequently, the disease frequently spreads from person to person, the drug would seem clearly to merit an extended trial.

In conclusion, if I may be permitted to do so, I would once again emphasise the great and probably preponderating *rôle* played by the urine in the dissemination of typhoid fever, and submit that we possess in urotropine a drug which is capable of rendering this excretion innocuous.

Dr. LONGHURST: Mr. President and gentlemen, I have nothing very new to add to the discussion, but having seen a good deal of typhoid fever both at home and abroad, I thought I would accept the invitation you gave at the last meeting of the Society to say a few words. In the first place, I endorse the opinions expressed by Dr. Tooth as to the great difficulties which attend all preventive measures in regard to the spread of the disease during a campaign. The whole circumstances of the life of the soldier, his surroundings, the climatic conditions, the torrents of rain, and other general circumstances, are all calculated to promote the disease and to conduce to its spread. I quite agree with him that we must look upon the urine as a very probable source of the spread in any campaign of this nature. The atmospheric distribution of the bacilli from this source, as well as from excreta, dust storms, and flies, becomes a most prominent factor in the extension of the disease. I am old enough to know the time when even the very nature of typhoid was under discussion, and when typhoid and typhus were put together, and conditions of slight fever were all classified together as Febris C.C. We have now arrived at a scientific position in which we know typhoid as typhoid; but at the same time we must allow that there are very many varieties, certainly many forms, of typhoid fever which are seen in different parts of the world, all varying materially from what we see at home. That has been my experience in India and Africa certainly. Cases of enteric in India were much more active and formidable in onset than in colder and more temperate climates. One of my observations has been that cold tends to the suppression of the disease; cold is unfavourable to the development of the germs, whereas moisture and heat tend to foster it.

A point which I think has hardly been dealt upon sufficiently to-night is the possibility that the typhoid bacillus may remain for a long time in the system in a comparatively inactive state; and then, as a result of certain atmospheric conditions, we may find that it is brought into activity much in the same way as in malarial fever. We see in every part of the world cases in which a man may be free for years, but for some cause or other he

suddenly gets an attack of ague or malarial fever. We must allow, I think, that in these cases it is not that he has been exposed to fresh atmospheric contagion, but that the bacilli have remained in his system. I suppose it may be the same with the bacilli of typhoid. I think those are very important points for us to consider.

With regard to the occurrence of relapses I think very much will depend on our own individual proclivity, or, on the other hand, our resistive power to the invasion of the disease. There is no doubt that all of us who are engaged in this work are exposed to it. It was thought at one time that typhoid was only to be communicated through the stools, or water, or milk; but I think we all accept now the fact that it is communicable from the patient to the nurse, and so on. It is thus very important to take every possible care in the disposal of excreta, but in the course of camp life it is almost impossible to do it effectually.

There is one point in the matter of treatment which I will refer to, and there are others I should have liked to have spoken of if there had been time. The point was this. I was glad to hear from Dr. Tooth that the cases of typhoid which were treated in tents by themselves were all more successfully treated than the others. Holding the opinion which I do, and which has been very strong in my mind for years past, as to the infective nature of the disease from one person to another, I have long felt that we are not doing justice to our patients when we collect them together in a fever hospital. My opinion is that typhoid should be treated in a room by itself. I am aware that such an expression of opinion is open to the objection founded upon pounds, shillings, and pence; but what are they to a scientific body of men who are not only trying to treat disease under conditions most favourable to the sufferers, but also endeavouring to prevent it? I say, instead of building large hospitals in London, and treating a lot of typhoid patients together, and putting half a dozen different patients suffering from typhoid in various stages in one ward, they should be isolated. I maintain that by putting them together in that way we are subjecting the earlier cases to relapses. I am glad of having had the opportunity of expressing this opinion.

One other point with regard to treatment Dr. Tooth alluded to, and that was that many of the cases were treated without stimulants at all. I think that is very important for us all to consider. We know it is said to be the teaching of the schools that typhoid fever must be treated by stimulants in large quantity. Only the other day I saw a case of the disease in a fine healthy lad, on the eighth day, who had a good deal of hæmorrhage from the nose (already pointing to congestion of the cerebral sinuses), and I was told he was being given four ounces of brandy every day. I think if you give four ounces of brandy per day under such conditions, with a good wrist pulse, you must expect to get hæmorrhage

from the nose. I am very glad to hear that many of the cases have been treated without alcohol at all.

The PRESIDENT: I much regret the time of the Society has come to an end, and that there are one or two speakers yet who would have liked to offer some remarks, but I cannot call upon them at this late hour. I will ask Dr. Tooth to be good enough to reply.

Dr. TOOTH, in reply, said: I think, Mr. President and gentlemen, the less I say at this late hour the better. Enteric fever has such a very large and general interest, being moreover a disease of which everybody has some personal experience, that it is almost impossible in a discussion of this sort to limit its range. In my paper I endeavoured to keep on the lines which you, sir, and your Council wished me to do; I therefore did not touch upon symptomatology. But as several points have been raised in the course of this discussion, I will refer to them in passing.

Surgeon-General Jameson spoke about the office of sanitary officer, which has been abolished. Of course any remarks that I may make on that must be taken as those of an amateur, that is to say, in questions of army medical work. I think there is room for sanitary officers, and perhaps I might even suggest some such corps as a Sanitary Corps. I agree with Sir William Church that on the march such a person might not be of very much use; but I am sure that a sanitary officer at Bloemfontein would have been of very great use indeed. Still such an officer must be invested with very considerable powers. He must be able to go to the very highest military authorities, and be able to speak very plainly to them. In the matter of shifting camps, for instance, it is obvious that a sanitary officer who considers such a move necessary must be able to back his opinion by the weight of authority. This of course is a question for the Army Medical Department to consider.

I agree with Surgeon-General Jameson and several other speakers when they refer to the extraordinary predilection that the soldier has for dirty water. No matter what pains were taken to boil or filter the water the soldier would still go to the drift with his own and his friend's bottle.

Professor Wright, whom I do not see here this evening, spoke of adjusting the dose of the toxine in inoculation. I suppose that the subject has advanced since he brought out the brochure which he sent out with the material. His directions then were—and I followed them very closely—that the ordinary sterilising process should be used, and then 1 c.c. of the toxine from a particular bottle, the exact dosage being usually put on the bottle. The dose was based upon the quantity which would be lethal to a certain weight of guinea-pig. That dose was used in every case; but though you may inoculate, from the same bottle and with the same quantity, a considerable number of men, the results will be very various. In some there will be a general febrile movement, and a

well-marked feeling of illness; in others there will be a little local pain at the spot of inoculation, and little else. Professor Wright speaks of adjusting the dose; but I do not see how one is to know beforehand how the particular person is going to react to the toxine.

Many interesting remarks fell from Major Macpherson; and several speakers have alluded to the flies and the particular breed which disseminated the fever. As far as I could see the fly that proved such a pest differed in no respect, as regards appearance at any rate, from the ordinary house fly.

I endorse what Sir William Church said about the way in which the flies pick out enteric patients. At Modder River, on going round the Guards' field hospital, one could pick out the men who were suffering from enteric by the number of flies upon their faces. There were men lying about equally ill with sunstroke, dysentery, and other diseases, but the flies scarcely touched them comparatively, but showed a marked preference for the enteric cases.

A very important point referred to by Major Macpherson was, that troops on the march do not suffer from enteric to the same extent as in camp. That suggests an important procedure which, it seems to me, was not sufficiently acted upon at Bloemfontein, namely, that camps should be repeatedly shifted. I remember an artillery siege-train camp which remained for a very long time in one place, and officer after officer was brought into our hospital. They reported that their camp was surrounded by smells, and one may speculate whether the smells from decomposing bodies and such like might have some contributory effect in increasing the virulence of the toxine. The latrine arrangements were on the whole good. But though in the daytime it is possible to see that the men make proper use of the latrines, it is not so after dark. It is scarcely surprising, therefore, that men will prefer to go anywhere rather than walk in the cold some two hundred yards to the latrine. When we bear in mind the probability of a considerable number of "ambulant" cases in a camp of some 20,000 men, the infective nature of the urine, and the dryness of the air, it is obvious that bacilli must be frequently deposited on the sand and then blown about by the wind.

Sir William Broadbent has made interesting suggestions about varieties of enteric fever. I must say from my own experience out there, at any rate on our side of the campaign—that is, in the Orange River Colony—that though there were some very bad cases, the type of disease was on the whole very much the same as that in England. Of course at the very height of the epidemic at Bloemfontein we did have some horrible cases. There was a particular batch of bad cases sent into the Portland Hospital from the 12th Brigade field hospital, which latter had to move when the general advance took place. I have never seen such terrible cases. They suggested to one's mind men who were suffering from septicæmia rather than

enteric fever. We made *post-mortem* examinations on all our fatal cases except three; and although they showed the specific ulceration of enteric fever, and there was no question about the diagnosis, some were so like cases of severe septicæmia that we used anti-streptococcic antitoxine; but I cannot say it ever did good. The virulence of the disease at that time was very great in many cases, but there were also a great many very mild cases, and the mortality, as Colonel Crombie has pointed out, is low; and I believe when the mortality returns are published the figures will come out extraordinarily low for the whole war. In our hospital, in which were a large number of selected cases, the total mortality was not high, namely, 12·5 per cent. For some years at St. Bartholomew's Hospital in London the mortality has been 15 per cent.; and at the Asylums Board Hospitals the rate has been sometimes as high as 21 per cent.

As to the question of thirst which Sir William Church spoke so feelingly about, the Guards medical officers told me that the thirst was so frightful at times on the march after fighting the battle of Belmont, and so on up to Modder River, that even they, who knew well what they were doing, and what the consequences might possibly be, were unable to restrain themselves, but would go and drink out of any puddle that they came across. Of course the soldier would care so much less, and it was curious to see how they would allow their mules and horses to get to the water first to stir it up, and then drink of it themselves.

I can bear out Colonel Crombie's remarks as to the frequency of thrombosis; it was certainly common in South Africa, and one saw and heard of a large number of cases in which this complication occurred. Coming home on the "Canada," we of the Portland Hospital took charge of the hospital on the ship, and on that vessel a large number of the cases were convalescing enterics. I should say that considerably more than half of those had thrombosis of veins of the leg, mostly on the left side.

There is a point which occurs to one in connection with enteric cases in a campaign like this. It is usual to send home any man who has suffered from enteric, but it sometimes happens that those who have had the disease lightly are allowed to go back to the front. I do not think that that is at all the right thing to do; we do not know how long the enteric bacillus persists in the body. We know it exists in many organs, and that it has been discovered to be present in the body a long time—in cases as much as two years—afterwards. Therefore I think it should be made an invariable rule that any man who has had enteric, however lightly, should be sent home, and should not partake any further in the campaign. That was brought to my mind by the fact that one of my patients at Bloemfontein, who had had two or three relapses, and was one of the worst cases, eventually recovered so far as the enteric was concerned, and was sent to the base. To my sorrow I

heard afterwards that he had gone back to the front and been shot. That was hardly fair on the man who had suffered so much, and it was not fair on his comrades, because a man so sent back takes with him the possibility of spreading the disease.

Captain Ward mentioned the sudden onset of the disease. In a considerable number of cases the onset was very sudden. I have wondered whether it was possible in some cases that the patients might have suffered from sunstroke first, for the initial stage was very much like that of sunstroke. I believe it is common in tropical and subtropical climates to have enteric fever beginning quite suddenly. These remarks apply rather to the Natal district, which is perhaps more than subtropical, and to the early cases at Modder River, where the heat was very great. In the Orange River at the time we were there it was far from tropical, for it was very cold at nights, and temperature in the daytime was like that of an ordinary English summer day; and as a rule the larger number of the cases at Bloemfontein began in the ordinary insidious manner.

Dr. Hillier raises some very interesting points, but as they rather trench upon matters of which I was careful to steer clear, I will not say anything about them. But he also brings in the question of the diagnosis of mild cases; this necessitates the consideration of the condition known in the army as "simple continued fever." That is a bone of contention, and is likely to be so for many a day. We had a number of cases sent down to us as "simple continued fever" when we were at Rondebosch. Many of those I believe are fevers of exhaustion; a man has a long march, is very much knocked up, has a fever lasting three or four days, gets over it, and is sent back. Sometimes by the time he gets to the base all the fever is gone, and there is nothing but a name. I believe a certain number of them are cases of diarrhœa or dysentery. Sometimes in cases of dysentery fever is a predominant symptom. Often in a case of apparently simple diarrhœa the thermometer shows a continued fever, which suggests a possible absorption of toxine. A considerable number of the cases of "simple continued fever," as we saw them, were undoubtedly enterics, and when we used the Widal test—and that is the only way to decide the point,—we found that they showed the reaction. Dr. Dodgson is quoted in the 'British Medical Journal' as working in the Raadzaal at Bloemfontein, and applying to a large number the Widal serum test. He found that, of the "simple continued fever" cases, 75 per cent. were enteric.

What Dr. Horton Smith says about urotropine is very important, and the drug should be used, if not in all cases before leaving hospital, at any rate in those in which the peculiar condition of urine that he describes is seen. But unfortunately this leaves untouched the ambulant cases which constitute such a danger of infection in crowded camps.

I find I have already much exceeded the time allowed, and will therefore conclude by expressing my thanks to those who have taken part in the discussion.

The PRESIDENT.—I am sure the Society feels greatly indebted to Dr. Tooth, and to the several speakers who have addressed us to-night, for the very interesting and practical discussion which we have had, and which I am sure has been very profitable to us all. I will now invite members to inspect the model of Lord Dundonald's water-cart.

The proceedings then terminated.

XIX.—*A case of Volvulus of the Ileum with severe Intestinal Hæmorrhage.* By J. H. Bryant, M.D. *Read April* 12, 1901.

I HAVE brought forward this case to-night on account of the great rarity of hæmorrhage *per anum* with any form of acute intestinal obstruction except intussusception. The following are the notes of the case:

Wm. O., æt. 21, was admitted under my care into Stephen Ward, Guy's Hospital, on September 18, 1900, for pain in the abdomen. He had always enjoyed very good health, and had felt quite well until about an hour or two after taking his supper on the 17th, at which meal he ate a large amount of damson pudding. At about 10 P.M. he was suddenly seized with severe pain in the umbilical and hypogastric regions, and as the pain became worse he came to the hospital just after midnight, and was admitted.

On admission his pulse was 80, the temperature 99°, and the respiration rate 24. He appeared to be in great pain, and rolled about from side to side and groaned. The pain was distinctly paroxysmal in character.

The abdomen was not distended, and it moved freely on respiration. The lower part was rigid. Just below the umbilicus and to the left of the middle line a hard mass of indefinite outline, about 5 cms. by 2·5 cms., was felt by the house physician, and below this and a little to the left of it—in the hypogastric region—another ill-defined mass was also made out, which was dull to percussion.

During the day the pain continued, and the lower part of the abdomen became tender. In the evening at 8 o'clock he became very collapsed, and passed about a pint of bright red blood, and a large mass of undigested damson skins about the size of a tennis ball. He became still more collapsed after this, and I was sent for and saw him for the first time at 9.15 P.M. I found him in a very collapsed condition, and he had a markedly blanched appearance. His pulse was 160, and it was feeble and running in character. He continued to pass blood in small quantities all through the night, and he vomited a good deal. The abdomen was not distended, Below the umbilicus, especially in the hypogastric region. the abdominal wall was very rigid, and it appeared to be

extremely tender, but no definite tumour could be made out. Nothing abnormal was felt *per rectum.* Injections of strychnine and infusions of normal saline solution were ordered, an exploratory operation being out of the question on account of his general condition. At 11 A.M. on the 20th the lower part of the abdomen was still rigid, hard, and tender, and he appeared to be in great pain. As he had not voided any urine for some time a catheter was passed, but only a moderate amount was found in the bladder. During the day no more blood was passed; in fact, nothing was passed *per rectum.*

At 2 P.M. he again became very collapsed and more saline solution was injected, a pint and a half being infused into his left axilla. He became very restless and delirious, and on account of the pain injections of morphia were ordered. Later on in the afternoon he was troubled a good deal with vomiting. At 11 P.M. he was restless, and another injection of morphia was given. He, however, gradually became more and more collapsed, and died about 5.30 A.M. on the 20th. Just before death took place a quantity of fæculent liquid poured from his mouth and nostrils.

I made a *post-mortem* examination nine hours after death. The body was well nourished and rigor mortis was well marked. The brain was not examined. There was no pleurisy. The lungs were normal.

The heart weighed 280 grammes. The myocardium and cavities were normal in appearance, and so also were all the valves with the exception of the aortic, the adjacent edges of the two posterior cusps being a good deal thickened, and firmly adherent to each other. There was no evidence of any recent endocarditis, or of old vegetations which might have become loose, or any rough or irregular surface from which an embolus might have become detached, or of any ante-mortem thrombosis in the left auricle or ventricle. The blood-vessels were normal.

The abdomen was a good deal distended. On opening the peritoneal cavity several coils of ileum of a deep dull red colour were found lying in and just above the pelvic cavity. The peritoneum had a dull and lustreless appearance, and was covered with large flakes and patches of recent lymph, which were loosely binding the affected coils of intestine together. The portion of intestine involved was found to be 100 cms. of the ileum, the lowest portion being 10·5 cms. above the ileo-cæcal valve; this affected portion of the gut was found to be attached to a long piece of mesentery which

had become twisted one and a half complete turns in its longitudinal axis.

The gut at the point of torsion did not appear to be completely obstructed, but the blood-vessels were markedly

FIG. 16.

constricted and strangulated. The intestine above the volvulus was distended, but that below was not collapsed. The wall of the affected portion of gut was much thickened, especially the part nearest to the mesentery, and it had a distinctly

œdematous feeling when pressed. The twist appeared to be from left to right. There was quite a pint of blood-stained fluid in the peritoneal cavity. There was no invagination of any portion of the intestine. I looked carefully, but could find no evidence of embolism or of thrombosis of the mesenteric vessels.

The liver was normal and weighed 1237 grammes, the spleen was normal and weighed 64 grammes.

The kidneys were normal and weighed 278 grammes.

The supra-renal capsules were normal.

Remarks.—The point of greatest interest in the case I have just recorded was the occurrence of a severe hæmorrhage *per anum* associated with a volvulus of the small intestine. Hæmorrhage from the intestine with any form of intestinal obstruction but intussusception is of exceptional rarity. Treves, in his article on "Intestinal Obstruction," in the third volume of Clifford Allbutt's *System of Medicine,* writes, "I have met with two or three recorded instances where blood is said to have been passed in acute cases other than intussusception." The same author in his monograph on intestinal obstruction mentions that Naunym reports a case of volvulus of the small intestine, in which much blood was passed *per anum.*

During life the presence of volvulus was not suspected. The patient was admitted as an ordinary case of *colica ab ingesta,* and the house physician did not consider his condition to be of sufficient gravity or urgency to request me to see him, until the sudden collapse which occurred after the hæmorrhage. When I first saw him he was desperately ill, and looked as if he could not survive for many hours. From the slight physical examination which was possible I was able to make out that there was no longer any definite tumour to be felt, but in its place there was some rigidity of the abdominal parietes, and considerable tenderness, suggesting a localised peritonitis. When I saw the evacuation, which consisted of a large mass of undigested damson skins moulded into a hard lump as large as a tennis-ball, and lying in the midst of quite a pint of dark red fluid, I thought it sufficient to explain the presence of the tumour which had been previously detected.

The dark red fluid in the bed-pan looked very much like damson juice, but it gave the guaiacum reaction, and on microscopical examination proved to be blood. I examined the rectum carefully, but there was no local cause for the

hæmorrhage, no hæmorrhoids, no ulceration, no neoplasm, and no intussusception. The tumour which was felt when he was first admitted suggested the possibility of intussusception, but its position was against this view, and also the fact of its disappearance after the mass of damson skins had been passed. Ulceration of the mucous membrane and acute enteritis from the irritation of the undigested damson skins was suggested as an explanation, but the gravity of the condition required something more than this to explain his symptoms. I thought of the possibility of ambulatory typhoid fever, but the absence of fever and other symptoms, and the very emphatic statement of the patient that he was in perfect health before eating his supper on the 17th, made this view untenable. On the whole, I was inclined to the diagnosis of thrombosis or embolism of the mesenteric vessels.

The next point of interest is the causation of the volvulus. The loop of the ileum was twisted about its own mesenteric axis, and the mesentery of the part involved was certainly longer than normal. I consider that the mass of damson skins must have played an important part in its production, the increased peristaltic movements of the ileum in order to force this foreign body through the ileo-cæcal valve causing the gut to become twisted about the pedicle formed by the abnormally long mesentery. The approximation of the ends of the loop, and the formation of the pedicle may have been aided by the weight of the mass of damson skins dragging down the coil of ileum. This view of the pathology is borne out by the fact that the presence of gall-stones in the intestine may cause rotation. Mayo Robson has reported two cases of this nature in the *Transactions* of the Royal Medical and Chirurgical Society, 1895, p. 117.

XX.—*A case of Ureteral Calculus simulating Vesical Calculus.* By Francis J. Steward, M.S. *Read April* 12, 1901.

CHARLES C., æt. 24, was admitted into Guy's Hospital under my care on August 9, 1900, for painful micturition and hæmaturia. On several occasions during the last three years the patient had slight attacks of painful micturition, but these had always passed off in a few days without treatment. Latterly, however, the pain had become more frequent, and also more severe.

These attacks were always accompanied by increased frequency of micturition, and also by the passage of a small amount of blood with the urine. The patient felt quite certain that he had never passed any stones.

A week before admission pain became much more severe, and the urine became scarlet in colour from the presence of blood, and continued so until admission into the Hospital. On admission the urine was acid, had a specific gravity of 1025, was quite red from the presence of blood, and also contained a small amount of pus. Micturition was accompanied by severe pain both in the perineum and also at the end of the penis. There was also pain at the end of the penis quite apart from micturition, but increased by micturition and also by taking exercise. The severity of the pain, the patient said, was such that he had been obliged to give up his work and seek advice. Micturition was also not only frequent but difficult, for he made frequent attempts without any result. Nothing could be made out by examination of the abdomen or rectum, and there was absolutely no tenderness or fulness over either kidney. It was thought that there was probably a stone in the bladder, so a sound was passed, but nothing could be felt. The patient was radiographed on two occasions with a negative result, and the kidneys and ureters again carefully palpated, but not the slightest tenderness could be elicited. The amount of blood in the urine gradually lessened after admission, as did also the pain to some extent; this was probably due to the fact that the patient had been kept in bed since admission. Feeling sure that there must be either a growth in the bladder or an encysted calculus, I decided to open the bladder above the pubes.

First operation, August 24.—The patient having been anæsthetised with the A.C.E. mixture, a soft catheter was passed and the bladder emptied. Twelve ounces of filtered air were then passed into the bladder, and the catheter clamped with forceps. A median incision 3 inches long was then made above the pubes. On separating the recti the inflated bladder immediately bulged into the wound, so that it was opened quite easily without opening up the cellular tissue in any way. On examining with the finger I found nothing in the bladder, but immediately felt a stone in the right ureter, through the bladder wall. The stone was situated at a point about 2 inches above the vesical orifice of the ureter. I tried to work the calculus down the ureter towards the orifice in the hopes of being able to extract it in that way, as has been done several times. I was, however, unable to move the stone at all, and so I closed the wound in the bladder with a fine continuous silk suture, and then sewed up the skin incision, with the exception of the lowest half inch, through which a small gauze drain was passed. A catheter was kept in the bladder for three days and then removed. No leakage took place, and the wound healed rapidly.

Second operation, September 4.—I decided to explore the ureter by opening the peritoneal cavity, since the stone was impacted so low down. An incision about 5 inches long was made over the outer edge of the right rectus in its lower part. On exposing the rectus muscle it was pushed inwards, the posterior layer of the sheath divided, and the peritoneum opened. The calculus was easily felt in the same position as before. I then proceeded to push the stone up the ureter, with the intention of removing it through a small lumbar incision, as Lane did in his case. The ureter, however, was not very much dilated, and it was only with some difficulty that the calculus could be moved, and it could not be pushed beyond a point just above the iliac artery. It was, therefore, decided to remove the calculus through the peritoneum; the intestines having been packed away with sterile gauze, and the edges of the wound well retracted, the peritoneum was incised a little internal to, and then stripped off the surface of, the ureter. A small longitudinal incision was then made in the ureter and the calculus extracted. At the moment of extraction about ʒj of urine escaped; this was quickly sponged away, and no more escaped. The ureter was closed with a fine continuous suture of No. 0 Chinese twist passing

only through the outer coats. The peritoneum was then closed over the ureter in the same way. A small gauze drain was passed from the lower angle of the abdominal incision down to the site of operation, in case any leakage should occur, and the rest of the wound closed. No leakage of urine took place, and the patient recovered quickly. A small stitch abscess formed at the upper part of the abdominal wound, and somewhat delayed convalescence. This I feel sure could not have been due to leakage from the sutured ureter, since the pus was immediately under the skin of the upper part of the wound, and did not reach so far down as the position of the gauze drain.

After the operation the blood in the urine steadily diminished in amount, and on September 8, the fifth day after the operation, ceased altogether.

From that time there has been no return of pain and the urine has remained quite normal. The patient was last seen on January 8, 1901, when he was in good health.

The calculus weighs nine grains, and consists probably of uric acid with a thin coating of phosphates on some parts. In shape it is a short cylinder, and is about half an inch long by a third of an inch in diameter, the surface being irregular and covered with small knobs.

The interest of this case lies chiefly in the peculiarity of the symptoms. For although it is well known that a calculus impacted at the vesical orifice of the ureter may give rise to symptoms pointing to vesical calculus, I have not been able to find any case in which a calculus impacted some way up the ureter has caused symptoms so typical of vesical calculus as this one did. For there was never a single symptom or sign pointing in any way to a unilateral lesion, although after the failure to detect a stone in the bladder with a sound most careful examinations were repeatedly made.

In this particular case the correct diagnosis was happily arrived at on exploring the bladder. Had the stone, however, been impacted a little higher in the ureter it could not have been thus felt from the interior of the bladder, and the diagnosis and treatment would have then become much more difficult.

Cystoscopic examination might have shown the blood to come from the right ureter had it been employed, and might lead to the diagnosis in another similar case, but failing this, the systematic exploration of each kidney and ureter would be the only means left.

The knowledge that symptoms so typical of vesical calculus may be due to a calculus in the ureter may possibly be of some help in dealing with similar cases, and it is for this reason that I bring this case to your notice.

With regard to the treatment there are two points which seem to me to be of some interest.

In the first place with regard to the value of air distension of the bladder. It appears to me to have all the advantages claimed for it. No distension of the rectum is necessary; the bladder becomes very prominent and makes itself immediately apparent on separating the recti, and no dissection and disturbance of the cellular tissue in the Cavum Retzii is necessary. Moreover, the absence of the gush of fluid on opening the bladder is another distinct advantage, since it prevents any possibility of infection of the surrounding cellular tissue.

I measured the air in this case and used twelve ounces, but in a more recent one I made use of Tilden Brown's suggestion, and made the incision down to the transversalis fascia before distending the bladder; in this way the exact amount of distension necessary to efficiently raise the peritoneal reflection could be easily gauged.

With regard to the method by which the calculus was removed I adopted the transperitoneal plan, because the calculus was impacted so low down that I doubted the feasibility of reaching it by an extra-peritoneal incision, although the latter would doubtless have been the safer plan in the event of leakage taking place.

The suturing of the ureter and the peritoneum over it was greatly facilitated by the use of Lane's cleft-palate needles and needle-holder, for the wound was so deep that accurate suturing by any other means would have been attended with considerable difficulty.

XXI.—*A case illustrating an attempt at the Antenatal Treatment of Hæmophilia.* By W. H. B. Brook, M.D. *Read April* 12, 1901.

THE following case may be of interest as an attempt at the antenatal treatment of hæmophilia.

On June 19, 1900, I was consulted by a lady æt. 34, who wished me to attend her at her confinement, which she expected early in October. Her family history showed a marked tendency to hæmophilia. A brother of her mother died at the age of eleven years from hæmorrhage following the extraction of a tooth. Of the patient's brothers, one died at the age of twelve years from gangrene following hæmorrhage into the knee-joint. Three brothers are alive and well. Four sisters are married; all their male children are well.

The patient's elder son, born in 1891, is a typical "bleeder," having suffered repeatedly from hæmorrhage into the joints, both knee, elbow, and ankle, and from bruising. During the second dentition his life was several times in danger from hæmorrhage from the alveoli, and he has twice suffered from hæmaturia without any apparent cause, the first attack lasting for four weeks, the last, in January of this year, lasting for two. At his birth he was a very white child, and there was much oozing of blood after the separation of the umbilical cord.

The patient's second child, a male born in September, 1894, was also very white at his birth, and there was also much oozing after the separation of the cord. He showed marked bruising at the age of three months, and died at the age of twelve months from what was diagnosed as cerebral hæmorrhage.

At the birth of each of these children the mother had considerable post-partum hæmorrhage, and was very weak for some time afterwards. The patient is a tall, well-formed woman of a dark complexion, and with a normally developed thyroid gland.

Having regard to this history, the problem presented itself of whether it would be possible to counteract or prevent the hæmorrhagic tendency by any treatment of the mother during the remainder of her pregnancy, and I was encouraged

to attack this problem by the perusal in the *British Medical Journal* for June 16, 1900, of two lectures on "Antenatal Diagnosis," by Dr. J. W. Ballantyne, Lecturer on Antenatal Pathology in the University of Edinburgh, with whom I communicated, and who most kindly interested himself in the case. With this end in view the patient was placed under the following treatment. Bearing in mind its power of increasing the coagulability of the blood, gr. x of chloride of calcium was given three times a day in the form of ♏ 50 doses of the Liquor Calcii Chloridi, without intermission up to the time of delivery. Further, with the view of improving the nutrition and tone of the vascular system of both the mother and fœtus, iron, strychnine, and arsenic was given in the form of a pill containing Pil. ferri gr. iv, Acid. arsen. gr. $\frac{1}{50}$, Strych. gr. $\frac{1}{60}$, three times a day up to September 17, when drachm doses of the syrup of the phosphates of iron, quinine, and strychnine were substituted, and this was continued until the time of delivery. The patient was encouraged to spend much time in the open air, and the bowels were carefully attended to.

Labour began on October 3 at 4 A.M., and a male child was born at 10 A.M., forceps being applied at the outlet. There was no post-partum hæmorrhage, and, indeed, very little loss at all. The umbilical cord was not tied until five minutes after the birth of the child, which was of a good size and of a healthy mottled appearance. It was not nursed. The patient made an excellent recovery without any untoward symptoms, and felt none of the weakness from which she had suffered after her two previous confinements. The child went on well; he was vaccinated on December 5, this causing no hæmorrhage. At the present time he is in excellent health.

I have ventured to bring this case to your notice, believing that, although a solitary one, yet it may be of some interest, as I have not been able to find any record of an attempt at the antenatal treatment of hæmophilia, but at the same time I am deeply conscious of its many weak points, namely,

First, the possibility that the child may yet show signs of hæmophilia, although he is at present healthy and strong. On the other hand, however, his second brother showed these signs at an earlier age, and if we may look upon the oozing after the separation of the umbilical cord as indicating a tendency to bleed, then the subject of this treatment is undoubtedly a healthier child than were either of his brothers.

Secondly, we have to bear in mind the possibility that, if this child goes through life without exhibiting any undue tendency to bleed, this may be merely a chance and not be due to any treatment. This is an objection that can only be finally decided by observing the success and non-success of treatment in other cases, and it is with this end in view that I have been encouraged to bring this case before you, in the hope that similar treatment might be tried by others.

Thirdly, we have a difficulty in believing that we can by any treatment by drugs alter a hereditary tendency; still I think that we may hope for this, having regard to the value of antenatal treatment in cases of syphilis.

Whether they are, or are not, the result of the treatment, two points are, I think, noticeable—namely, the fact that whereas at her two previous confinements the patient had had much post-partum hæmorrhage and had suffered long from weakness, this time there was no post-partum hæmorrhage and no weakness; and secondly, the fact that whereas the two former children at their birth were white and weak, this one was healthy, strong, and of a good colour.

XXII.—*Poisoning from the External Use of Aniline Oil.* By StClair Thomson, M.D. *Read April* 12, 1901.

An article appeared in the *Lancet* of April 21, 1900, on the subject of "The Production of local Anæsthesia in the Ear," by Dr. Albert A. Gray. Therein the writer referred to the fact that as yet no satisfactory local anæsthetic had been discovered for ear affections, and he detailed some experiments he had made with the object of finding a vehicle which would penetrate the tissues rapidly without destroying them. He found that alcohol was absorbed too rapidly, but that a mixture of aniline oil and rectified spirits met the case admirably. To this he added 5 to 10 per cent. of cocaine hydrochlorate. Ten to fifteen drops of the mixture of equal parts of aniline oil and spirit may, he says, be dropped into the ear and left there in the usual way once or twice a day. In practice Dr. Gray had always filled the external meatus with the fluid, and had never seen any serious effects of cocaine poisoning by so doing. In conclusion he states, on the authority of Professor Stockman, that the medicinal dose of aniline oil is seven minims, and that care should be taken that not more than this amount is absorbed, though in the ear such a contingency is very unlikely.

Impressed with this article I looked forward to an early opportunity of testing the value of an aniline oil solution of cocaine in ear disease. This soon presented itself in the case of a colleague who consulted me for uncomplicated furunculosis of the external auditory meatus. On the anterior wall of the meatus was a tense red painful swelling in an early stage; on the floor of the meatus was a softer furuncle which had "pointed" and was oozing a droplet of pus. The patient was not anxious to have the furuncles incised, and I gave him a prescription for equal parts of aniline oil and rectified spirits to which 10 per cent. of cocaine hydrochlorate was added.

A small pledget of cotton wool moistened with this solution was introduced into the painful ear that evening, and the patient attributed decided relief to the application. He slept well, but next morning, as the pain threatened to return, he again made use of the drops about 5 A.M. At 7.30 A.M., while still in bed, he quite accidentally noticed a peculiar blueness

of his finger-nails, and as his wife observed that his face was also blue she sent for me. I reached the patient's house at 9 A.M. and found him perfectly calm, cool, and collected. There was no excitement or fever, no apparent anxiety or distress, and he gave a clear account of himself, complaining only of a sense of prostration. His face and hands were of a decided dark blue colour. This was most noticeable in the skin under the finger-nails, and on the lips and tongue. By contrast the conjunctivæ appeared abnormally pale.

The appearances suggested to me a similar condition I had seen from an overdose of antipirine, but there was no history of any drug having been taken. The symptoms were entirely different from those produced by cocaine intoxication. The pupils were normal instead of being dilated. The patient was quite calm and self-possessed, instead of being excited, restless, and loquacious. The face was blue instead of pale. The respiration was quiet and easy, instead of being sighing, irregular, and hurried. And, finally, the pulse although small and somewhat more frequent was not so rapid and soft as in cocaine poisoning.

There was nothing in the condition of the patient's ear to account for the symptoms, of which the marked blueness was the most striking and alarming. I therefore simply ordered strict rest and a strong cup of black coffee.

Dr. Lees, who had been summoned, arrived shortly afterwards, and found that the dulness of the left ventricle was enlarged to two finger-breadths outside the left nipple line. There was no dyspnœa. The patient had not previously had any heart trouble. There was nothing discoverable in the heart or lungs to account for the cyanosis, and it was doubtless due to the toxic effect of the aniline oil on the red corpuscles. The urine was free from albumin.

The patient was kept in bed all day, the blue colour gradually disappearing, and by next morning no trace of it remained. Dr. Lees examined the heart a fortnight later, and found that the area of cardiac dulness had again become normal, and that no murmur was discoverable.

The above observation was made in the month of June, 1900. On July 17 a communication was made to the Academie de Medicine by MM. Landouzy and Georges Brouardel on "Non-professional Poisoning by Aniline."* Ten cases had come under their notice in which children, soon after wearing yellow shoes which had recently been coated

* *La Presse Médicale*, 18 Juillet, 1900.

with a black tincture, were seized with prostration, pallor, and blueness. Slight albuminuria was present in some cases. Investigation resulted in incriminating the dye which had been applied to the boots. It contained 90 per cent. of aniline. Experiments were made on animals (guinea pigs and rabbits) by applying a layer of the dye to a shaven surface as large as the palm of the hand, covering it with moist cotton wool, and fixing it by a bandage. The animals died asphyxiated in twenty-four to thirty hours.

These experiments justify two conclusions:—(1) The first is that the toxic symptoms exhibited by my patient were not due to any peculiar susceptibility or idiosyncrasy. (2) The second is that the skin readily absorbs aniline, provided it is covered and kept moist and warm. Now the external auditory meatus will always present the requisite of warmth; it will in many diseases offer the required moisture; and the only condition we can avoid is that of keeping covered any application of aniline.

In the cases recorded by MM. Landouzy and Brouardel all the requisites of warmth, moisture, and protection were supplied in the close-fitting boots. Any works which I have consulted with reference to the action of aniline oil are silent as to its toxic action when applied externally. Further observation or experiment appears necessary to determine whether its poisonous effects are produced when it is simply applied to the skin and not kept covered. I understand that since the appearance of Dr. Gray's article the combination of cocaine with aniline oil and rectified spirit is being extensively tried by aurists both in this country and abroad. The publication of the above case will, I trust, elicit further communications on the subject, and put practitioners on their guard against a possible occurrence which is alarming in character and might otherwise be inexplicable.

Postscript.—Since the above notes were sent in to the Society on January 19 last, Dr. Albert A. Gray has published a second article on the subject,* in which he mentions that his method has been widely adopted both in this country and abroad. Although he had not seen any symptoms of intoxication either by the aniline or the cocaine, he refers to two cases in which a little trouble resulted. One was evidently a case of cocaine intoxication. In the other case the solution was instilled into the meatus, and in the course of an hour or so the patient's lips became blue, and slight gastric catarrh

* *Lancet*, March 9, 1901.

occurred. He adds, however, that several of his patients told him that an hour or two after the use of the solution their friends noticed this peculiar blue colour. No symptoms were, he states, present in any of the cases, and the patients would not have known about it had their attention not been drawn to it by their friends. The condition passed off in the course of a few hours and left no effects. He suggests that its occurrence may be avoided, if so desired, by limiting the dose to twenty minims for adults, and corresponding doses for children.

Seeing the announcement of this contribution in the medical journals, Dr. Kelynack of Manchester has very kindly forwarded to me some unpublished notes of two cases of acute poisoning and three of chronic poisoning amongst those employed in aniline works. They confirm the possibility of absorption through the skin, producing not only the symptoms of blueness and cardiac depression, but also of gastro-intestinal catarrh and congestion of the bases of the lungs. In the chronic cases there was in addition marked anæmia. It is to be hoped that Dr. Kelynack will publish his observations in full, as they confirm the possibility of alarming symptoms being induced by the external use of aniline oil.

XXIII.—*A case of Cancer of the Stomach, with unusual vascular and blood changes.* By W. PASTEUR, M.D., and C. PRICE JONES, M.B. *Read May* 10, 1901.

ROBERT T., æt. 61, a metal chaser, was admitted to hospital on January 29 of this year. Except that an uncle had died from cancer the family history was unimportant. He had had from childhood an inguinal hernia which became strangulated in 1898 and was operated on. In 1878 he had rheumatic fever. He was not strictly temperate as a young man.

For the last five years he had been liable to gastric trouble, but it was only during the last two that he had been under more or less regular observation as an out-patient. His chief symptoms at this time were epigastric pain after food relieved by vomiting, and a tendency to constipation and flatulence.

Throughout this period he benefited considerably under suitable medicinal treatment, so that he was enabled on more than one occasion to interrupt his visits to the hospital for two or three months. When he reappeared in the out-patient room for the last time, after an absence of nearly five months, his appearance was much changed. He had lost flesh considerably, and was very weak and bloodless.

On admission, great pallor of the skin and mucous membranes, with marked emaciation and feebleness, were the most prominent symptoms. Vomiting was only occasional, and pain was less marked than during the earlier stages of the illness. The pulse was 120, soft, and compressible; respirations 24; temperature normal. Urine, sp. gr. 1013; acid; no albumen; no sugar. Numerous hard subcutaneous nodules about the size of a split pea were observed over the abdomen, and to a less extent over the chest and shoulders. They were painless and not adherent to the skin. There was some fulness of the epigastric and left hypochondriac regions, with a sense of deep resistance on palpation. To the left of the umbilicus there was a hard irregular mass which moved with respiration. Resonance was slightly impaired over it, but a normal stomach note was obtained just above. A

smaller mass of similar character was felt a short distance above this one. The liver was of normal size, its lower border being just perceptible on deep inspiration.

The patient made little if any complaint of pain, and only vomited once after admission. The vomited matter contained no free hydrochloric acid (Congo paper).

Two days after admission (January 31) the temperature rose suddenly to 100° F., without obvious cause, and fell again below the normal on February 3.

On February 4 the patient was weaker and had lost control over the rectum. It was then noted that the greater part of the nose had become dusky. The patient himself was, however, quite unconscious of any unusual sensation in his nose.

On February 6—two days later—the nose was of a uniform deep purple colour and insensitive to light touches. The toes of both feet were similarly affected, presenting the blue-black appearance often seen in Raynaud's disease. The discoloured area ended abruptly a little beyond the base of the toes. The toes were warm but quite insensitive, and pressure with the finger did not appreciably modify the colour.

On February 7 there was slight œdema of both feet as far as the ankles. No pulsation could be felt in the posterior tibial or dorsal arteries on either side. The toes were now cold and insensitive and remained entirely free from pain. A bulla containing clear fluid had formed on the dorsum of the right big toe. (Cultures were made after death from the serum of this blister, but the tubes' broth, gelatin and agar, all remained sterile.)

An examination of the blood made on this day gave the following results:

Red cells,	1,460,000	per cub. mm. by Gowers and Toison's method.
White cells,	24,500	

Analysis of white cells (wet films stained by Jenner's method):

Small lymphocytes,	20·00 per cent.	44·59 per cent. of hyaline mononuclear cells.
Large ,,	13·33 ,,	
Large mononuclear cells,	4·60 ,,	
Transitional cells,	6·66 ,,	
Polymorpho-nuclear neutrophils,	50·76 ,,	54·35 per cent. granular polynuclear cells.
Eosiniphile cells,	3·59 ,,	
Basophile myelocytes,	1·02 ,,	1·02 per cent. of granular mononuclear cells.
Eosiniphile ,,	0·00 ,,	
Mast cells		None observed.

A large number of normoblasts, megaloblasts, megalocytes, microcytes, and poikilocytes.

February 9.—Appearance of nose and feet unchanged; a few additional small bullæ on the right foot. Hands not affected.

February 10.—Dyspnœa and deepening stupor. Death.

No visible changes occurred at the margin of the discoloured areas during life, and no hæmorrhages or ecchymoses occurred in any other part of the body. We may add that there were no variations in the intensity of the colour of the nose and feet from day to day.

Autopsy (twenty-five hours after death).—There was a purplish-brown discoloration of the skin of the nose, with sharply defined margin, which included the tip and alæ, and extended up as high as the nasal bones. No bullæ; no œdema; no abrasions. There was a similar condition of the skin of all the toes, front and back, extending for one inch on to the foot. The edge was not more deeply coloured, nor was the skin at the edge either raised, depressed, or œdematous. There were a few small bullæ on the right toes, which contained clear fluid. The right posterior tibial artery was patent and appeared healthy to the naked eye.

Thorax.—With the exception of fibrous scars at both apices, enclosing one or two small cretaceous masses, and some hypostatic congestion of the bases, there was nothing calling for mention in the state of the lungs.

The heart was pale and rather flabby (weight 10¼ ounces), and showed signs of fatty degeneration.

Abdomen.—The colon was distended. The peritoneum was healthy. The stomach was enlarged. Near the pylorus, on the lesser curvature, the stomach was infiltrated by a mass of new growth, which also extended backwards, involving the prævertebral glands. The pylorus was of normal size and unaffected. There was considerable infiltration of the tissues of the lesser omentum, which formed a hard rigid mass. The head of the pancreas was also infiltrated. The liver was slightly enlarged, but did not contain any secondary cancerous deposits. There was no indication of the deposition of iron in the liver on testing with potassium ferrocyanide and dilute hydrochloric acid, and likewise no result with potassium ferricyanide. The kidneys, adrenals, spleen, and brain presented normal naked-eye appearances.

Examination of Marrow (Jenner stain).

Marrow of Tibia.—White; consists of fat-cells and broken-down cells.

Marrow of Rib.—Pale yellow; consists of fat-cells; a few red cells; one normoblast only in one film; myelocytes few, small and basiphil; no eosiniphil myelocytes; some large broken-down cells. No thickening of bone of ribs.

Spleen.—Films from this organ show many lymphoid and red cells, numerous megalocytes, a few megaloblasts and normoblasts, and eosiniphil myelocytes.

Microscopical Examination of Cut Sections from the Stomach, Aortic Glands, and Subcutaneous Nodules.—The growth from the stomach and aortic glands is a glandular carcinoma, with a minimum of fibrous stroma. It is undergoing colloid degeneration. The subcutaneous nodules consist of almost pure colloid material, but few cells of the original growth remaining.

Remarks.—The two features of this case on which we have chiefly dwelt, viz., the condition of the extremities and the state of the blood, appear to us of sufficient interest to put the case on record.

The capillary stasis in the nose and feet was indistinguishable from that often met with in Raynaud's disease, and it is not unlikely that, had the patient survived longer, the resemblance would have been further strengthened by the supervention of gangrene. It is remarkable that the ears and fingers should have entirely escaped.

Regarding the state of the blood, it is to be regretted that only one examination was obtainable, and this four days before death. The cell enumeration by Gowers' method gave red cells, 1,460,000 per c.mm.; white cells, 24,500 per c.mm.

Films treated with Jenner stain showed many points of resemblance to the blood of pernicious anæmia, viz.—(1) A large number of megalocytes, poikilocytes, and polychromatophilic red cells—*i. e.* increased average diameter of red cells. (2) The presence of nucleated red cells. (3) A condition of lymphocytosis; but differed from the blood of pernicious anæmia, (1) by the increased number of white cells; (2) by the analysis of the nucleated red cells, which showed only 37 per cent. of megaloblasts, whereas, according to Cabot, in pernicious anæmia the megaloblasts should be in excess of the normoblasts.

Abnormal states of the blood-cells somewhat resembling

those occurring in pernicious anæmia have been recorded by various observers in cases of malignant disease: but the results so far seem inconclusive, and present no special diagnostic character. It is to be hoped that further investigations will, before long, throw more light on our knowledge of the blood changes in these secondary anæmias of chronic wasting diseases. We consider our case as belonging to this category, and believe that the condition of the marrow still further differentiates it from one of true pernicious anæmia.

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DESCRIPTION OF PLATE VIII.

To illustrate Dr. Pasteur and Mr. Price-Jones's Paper on Cancer of the Stomach.

The illustrations show the capillary stasis of the nose and feet.

From water-colour drawings by Mr. Price-Jones.

Fig. 1.

Fig. 2.

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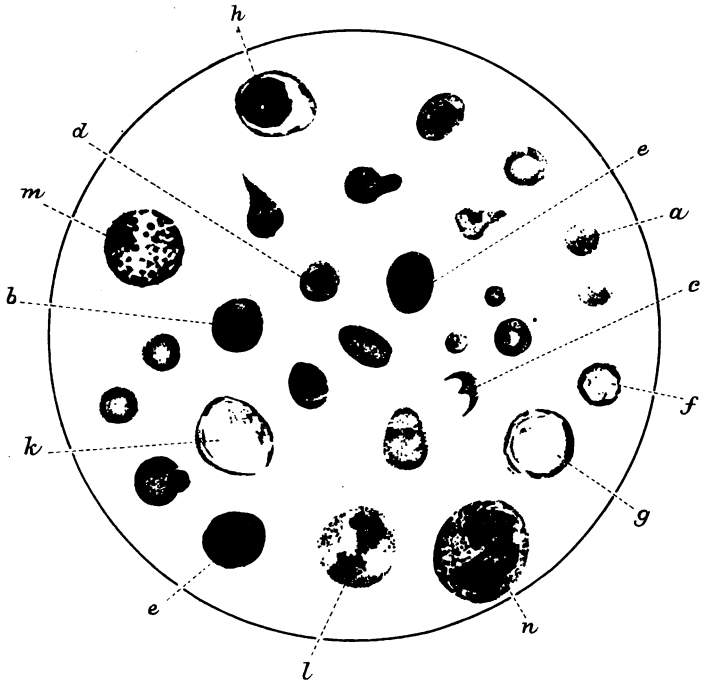
DESCRIPTION OF PLATE IX.

To illustrate Dr. Pasteur and Mr. Price-Jones's Paper on Cancer of the Stomach.

Red Cells.—*a*, **Normal cell.** *b*, **Megalocyte.** *c*, **Poikilocyte.** *d*, Normoblast. *e*, Megaloblast.

Hyaline White Cells.—*f*, Small lymphocyte. *g*, Large lymphocyte. *h*, Large mononuclear cell. *k*, Transitional cell.

Granular White Cells.—*l*, Polymorphonuclear cell. *m*, Eosinophile. *n*, Myelocyte.



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XXIV.—*A case of Perforating Gastric Ulcer, causing a subdiaphragmatic abscess, which burst through the diaphragm into the lung.* By NORMAN DALTON, M.D. *Read May* 10, 1901.

THE patient, F. R., a parlourmaid, æt. 25, was admitted into King's College Hospital on February 20, 1901. Her mother and a sister had died of phthisis. Her past history was unimportant, except for a single pregnancy some years ago, and slight dyspepsia of late.

On February 12, eight days before admission, she felt a slight aching pain below the cardiac area in the left mammary line. On the 13th this pain was worse. It was now stabbing in character, and was exacerbated by drawing a deep breath, but there was no cough. On the 14th there was retching, but no real vomiting, and no hæmatemesis. The pain was very severe during vomiting. On the 15th she remained in bed, the symptoms being as before. On the 16th she left her place, and went to stay with friends. I believe that the journey was only from one part of London to another. On the 18th she felt better and went out, but could not go far, as her breath was short. On the 19th the pain was as severe as before, and she went to a doctor.

It will be noticed that she had now been ill for one week, but without any symptoms except pain and dyspnœa. There had been retching one morning, but otherwise no stomach symptoms, and she took her food well. There had been nothing like collapse. We have no information as to any physical signs which may have existed during this week. Her doctor diagnosed pneumonia, and on the 20th she came into King's. The temperature was 102·6°, the pulse full and bounding, and the face flushed. There was dulness at the left base, and as high as the second rib in front, and, in fact, all the signs of fluid at the left base were present. In particular, the heart was displaced to the right. There was, however, one anomalous point to which I drew attention at the time; although the dyspnœa was very great, and the left side of the chest immobile, the patient lay on her right, *i. e.* on the sound side. This was due to the extreme pain which occurred when she turned over on to the left side, and so pressed the affected side against the bed by the weight of

her body. The pain was also increased when she sat up. It was therefore clear that the distress of the pain was worse than that of the dyspnœa, which was nevertheless very urgent. I think this symptom of great importance, and that it may be of use in future cases. It could not, of course, be taken to indicate a perforating gastric ulcer, but it should have suggested that the pain was greater and of a different character to that usually met with in pleurisy.

The left pleura was immediately aspirated, and fifteen ounces of fluid were drawn off. The fluid was slightly blood-tinged, but quite clear, without a trace of pus or fibrin. After tapping tubular breathing was heard in the left axillary region, where previously no breath sounds could be made out, and slight crepitation was audible at the left apex and at the right base.

The temperature and the character of the pulse suggested that there might be pneumonia beneath the effusion. On the other hand, no rigors had occurred, there was no cough and no sputum, and it is unusual for so much fluid to accumulate in the pleura in ordinary lobar pneumonia.

The diagnosis of pleurisy with effusion, possibly tubercular in origin, had to be adopted, but even then the absence of cough was remarkable.

For the next notes I am indebted to my house physician, Mr. W. Stuart. The breathing was relieved by the tapping, and she felt better, but the temperature did not fall appreciably. On the evening of the 21st there was a slight rigor, and on the morning of the 22nd a very severe one, after which the temperature rose to 106·2°. It fell in a few hours to 101°, but soon rose again to 104°, near to which point it remained for the next few days. No vomiting occurred, and the pain was not worse on taking food, so that there was nothing to suggest a gastric affection. I saw her again on the 23rd. The pain was now worse and the dyspnœa had returned. There was still no cough. Examination revealed a remarkable condition. The apex-beat was just external to the left nipple, *i.e.* it had crossed over from the right, and was now displaced upwards and to the left, which was the affected side. The dulness which had been observed before tapping was still present; in front between the heart and the second interspace, behind over the lower three quarters of the left chest, and in the upper part of the axilla; but the lower part of the axillary region and the area between the heart and the margin of the ribs was now tympanitic. No breath-

sounds were heard over this area. The tympanitic sound was exactly like the stomach note, and I thought that the lung had, for some reason, not expanded, and that the diaphragm had consequently risen, taking the stomach with it. The first part of this hypothesis was correct, but the second was erroneous, for the tympanitic area was really that of the cavity outside the stomach, which had formed round the stomach contents. The displacement of the heart to the left confirmed the idea that the left lung had not expanded when the fluid was withdrawn. The other physical signs were unimportant. Over the dull area of the left lung we heard frictions and distant tubular breathing; the heart's

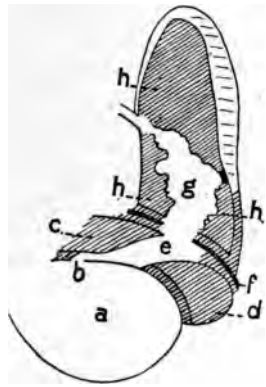


FIG. 17.—Showing the position of the affected parts. *a*, stomach; *b*, ulcer; *c*, liver; *d*, spleen; *e*, subdiaphragmatic abscess; *f*, diaphragm; *g*, abscess in lung; *h*, lung.

sounds were very feeble, and there was a doubtful pericardial friction, which was put down to the proximity of the pleura.

The case may now be said to have entered upon its second stage, for the rigors and fever suggested pyæmia. As the fluid which had been drawn off was clear, I thought that the disease had not been originally septic, but had now become so. The displacement of the heart to the left negatived the idea that the fluid had re-accumulated in the pleura in the form of pus, or that pneumothorax had occurred. The question of septic endocarditis was considered, and also that of infarction at the base of the left lung, caused by an embolus from the uterine or some other veins. Another idea which

was entertained was that a small foreign body had become impacted in a bronchus in the lower lobe of the left lung. Either an infarct or a foreign body might have excited a simple pleurisy at first, and have become septic afterwards. There was, however, no definite evidence in favour of any of these views, and the absence of cough and expectoration was against them all. Further, there were no crepitations at the affected base—only faint tubular sounds over the dull area, which might have been caused by non-suppurating pneumonia or by collapse.

The patient lived seven days longer, but until near the end there was no great change, except that the pain extended to the back, to a place corresponding to the top of the spleen. It was very severe, and she obtained greatest relief by lying on her face. Once or twice I thought I heard a systolic bruit at the apex of the heart. There was a rigor on the 24th, and two more on the 27th. After one of the latter the temperature rose to 107°. The fever was high and remittent all the time, but fell, temporarily, after sponging. The physical signs did not change much, except that the tympanitic area became a little smaller, especially towards the axillary region, while the apex-beat reached to the nipple instead of just to the left of it. No metallic chink with coins could be obtained over the tympanitic area. The greater part of the lung remained dull as before, with distant tubular breathing, and sometimes a little crepitation and frictions. The tongue became brown, but the mind remained clear.

On the morning of March 2 she was much worse. The heart and the tympanitic area were as before, but there were abundant crepitations over the dull area behind and also over the right base. She now for the first time began to cough and bring up sputum, which was muco-purulent, but contained no blood. It had a remarkably sour smell, like that of sour milk, which attracted our attention, and should have suggested a communication between the lung and the stomach. But she was now moribund. Pneumococci and streptococci were found in the sputum, but no tubercular bacilli. She died that night, but before death the sputum had become so gangrenous in smell that the whole ward had to be deodorised.

At the post-mortem the pericardium and the heart were normal, and the only lesions of importance concerned the left thorax and the stomach. The lower third of the pleura posteriorly was obliterated by dense masses of fibrin. This corresponded to the area from which the fluid had been with-

drawn, and the presence of the fibrin proved that the original lesion at that part had been a pleurisy and not a hydrothorax, which made the absence of cough the more remarkable. The upper part of the left pleura contained a few ounces of fluid, which, although so near to the suppurating lung, was not at all purulent. The surface of the lung in contact with the fluid was covered with fibrin, and at a spot on the posterior surface of the lower lobe, just above the fibrinous adhesions, there was an opaque, dirty-white area about the size of a crown piece. This proved to be a portion of the wall of the abscess in the lung. It was quite necrosed, but had not separated, so that there was no pneumothorax. The lower surface of the left lung was adherent to the diaphragm and the ribs in front, and while the sternum was being removed in the usual way the lung was torn, and an abscess cavity in it was opened. This abscess occupied nearly the whole of the

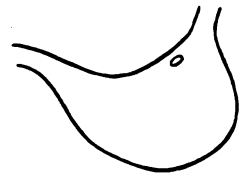


FIG. 18.—Showing the position of the ulcer.

lower lobe of the left lung, and contained air, dirty pus, and grey sloughs. The contents were horribly offensive. There was a ragged hole in the diaphragm which communicated with the pulmonary abscess above, and with another abscess below. The sub-diaphragmatic abscess contained the same stuff as the abscess in the lung, and was somewhat funnel-shaped, with the apex of the funnel below. Its upper wall was formed by the diaphragm, its left lower wall by the upper side of the spleen, and its right lower wall by the lower surface of the liver. From the apex of the funnel a flattened channel burrowed between the under surface of the liver and the anterior wall of the stomach. This channel led to the gastric ulcer. The abscess cavity was quite shut off by adhesions, and there was no general peritonitis, not even over the free part of the stomach. On opening the stomach an ulcer about the size and shape of an almond was found on the anterior wall, about 2 inches from the cardia, and close to the lesser curvature. The base of this ulcer was formed by a slough

(which had not completely separated) and by the adherent under-surface of the liver. It now appeared to be quite closed, but there can be no doubt that it had communicated with the sub-diaphragmatic abscess by means of the channel between the liver and the stomach. A second unperforated ulcer, the size of a sixpenny-bit, was found close to the first ulcer.

On reviewing the symptoms after the post-mortem, it was evident that the case followed fairly well the usual course of events which occur in cases of subphrenic abscess, and I can only hope that this full report may be of use to others. I would emphasise the following points which have been mentioned by writers on this complication, viz., that these cases may occur without any symptoms to draw the attention to the stomach; that the fluid in the pleura, although close to a foul abscess, may be quite clear, and that the lung symptoms may obscure the real state of affairs. I would also draw attention to the great pain in this case, especially when the affected side was pressed against. The absence of cough in this case is of less importance, because, after all, pleurisy was really present, and should have excited its own peculiar cough. The absence of sputum was not remarkable because there was no cough, but it is of the utmost importance to note that the first sputum expectorated (which, in this case, was about fourteen hours before death) smelt of lactic acid, and that in the course of a few hours this specific odour was replaced by an overwhelming gangrenous effluvium. This seems to indicate that, in a difficult case, there might be a fortunate period at which the smell might reveal the connection of the lung condition with the stomach, and if the patient were not, as in this case, moribund, such knowledge would bring about an operation which, even at the eleventh hour, might not be too late. But the rapid supervention of the gangrenous odour shows that the fortunate period would be brief. As regards mistaking the tympanitic area for the percussion note produced by a stomach which was drawn upwards by non-expansion of the lung, I will plead that the left border of this tympanitic area was curved in a way which suggested the greater curvature of the stomach, and that the heart was displaced to the left as well as upwards.

XXV.—*Some cases of Ascites treated by Abdominal Section.* By LEONARD A. BIDWELL. *Read May* 10, 1901.

I WISH to bring forward the following three cases as a small addition to the evidence in favour of the surgical treatment of ascites.

The operative treatment of ascites in connection with cirrhosis of the liver was first advocated by Dr. Drummond and Mr. Morrison, and more recently a valuable paper on the same subject was read before the Medical Society by Dr. Rolleston and Mr. Turner. In this last-named paper the means by which the operation may effect a cure are fully discussed, and so there is no need to enter into this question here. Out of my three cases, however, in only one was the ascites due to cirrhosis of the liver, in the second case the cause is still obscure, and in the third the cause was cardiac disease.

CASE 1. *Ascites due to cirrhosis of the liver in a syphilitic patient.*—Mrs. H., æt. 50, who had previously enjoyed good health, at the latter part of 1899 developed a peculiar specific eruption which Dr. Sinclair, of Parsons Green, asked me to see in January, 1900. This eruption quickly subsided under mercurial inunction, although it had been quite unaffected by iodide of potassium, and the patient regained her health. In March she developed a specific ulcer of the tongue, which also healed up after the application of acid nitrate of mercury in addition to the inunction. She then was practically well till the beginning of July, when the abdomen became swollen and ascites was found, the liver being much enlarged and irregular in outline. She was treated with Glauber's salts and mercurial inunction for about a fortnight without any decrease of the ascitic fluid. The abdomen was then greatly distended and the respiration was considerably interfered with, and as the patient appeared to be rapidly going down hill with a very weak and rapid pulse, an operation was proposed.

On July 16 the patient was anæsthetised by Dr. Shuter, and the abdomen was opened in the middle line above the umbilicus. More than a gallon of ascitic fluid was evacuated

and the enlarged liver was well scrubbed with cyanide gauze wrapped round the handle of a retractor. The adjacent parietal peritoneum was treated in a similar manner; the liver and the omentum were united to the parietal peritoneum by silk sutures; the intestines were also well rubbed with a sponge. The abdominal wound was closed by three layers of sutures.

The patient recovered completely from the operation; there was not any rise of temperature, but she had an attack of jaundice on the third day, and at the end of a week there was a slight collection of fluid in the abdominal cavity; this, however, rapidly decreased, and by the end of August had completely disappeared.

The patient was able to get about again, and never had any further ascites; she seemed to be quite well until December 17, 1900, when she had an attack of hæmatemesis; this recurred three times during the day, and the patient died of exhaustion on the same evening. On the previous evening she had indulged in excess of alcohol. No autopsy was allowed.

Case 2. *Attacks of albuminuria, general œdema, and ascites.*—In December, 1898, I was asked to see Master B., æt. 11, by Dr. Passmore, of Gainsborough, who gave the following history of the case. The boy had been quite well up to April of that year, when he developed acute nephritis with dropsy of the face, scrotum, and legs, the urine being loaded with albumen. Soon after this an abscess formed on the outer side of the left thigh; this was opened and drained, and the general condition of the child improved, although the albumen never completely disappeared. In July he had another acute attack of dropsy, accompanied by abdominal pain, and some swelling was found in the region of the left kidney. The dropsy and abdominal pain subsided and the albumen cleared up, but the boy had similar attacks about every month, and on one occasion he had suppression of urine. The last severe attack had occurred six weeks before I saw the child, but even then his face was puffy and there was albumen in the urine; there was not any pain or tenderness over either kidney.

In January, 1899, a tender swelling appeared in the region of the left kidney, and this swelling enlarged and filled the whole of the left side of the abdomen; ascitic fluid then began to collect. He was tapped for the first time on January 26, only a few pints being removed, and the swelling over the

left kidney being unaffected. He required tapping again on February 4. On February 13 the abdominal swelling burst at the umbilicus, at first about two pints of clear fluid escaping, followed by about the same quantity of thin puriform fluid. This latter fluid was examined at two laboratories in London, but no trace of tubercle bacilli or of hydatids were found. Although some albumen still remained in the urine the patient then improved, and seemed fairly well till September, when he had another acute attack of dropsy, accompanied by abdominal pain and by albuminuria. The abdomen again became distended, and he was tapped again on October 23; after this the fluid did not collect so quickly, and he did not again require tapping until December 17; from this time he remained free from any attacks of pain or of albuminuria for about six months. In July, 1900, the fluid began to re-collect, and there was general anasarca; he was tapped on July 24, and again on August 29. As the abdomen had refilled very rapidly, on September 2 the child was anæsthetised by Dr. Passmore, and the abdomen was opened on the right of the middle line at the level of the umbilicus. About six pints of clear fluid were evacuated, and the abdominal cavity was carefully explored; nothing abnormal was detected, with the exception of some matting round the left kidney, and the liver was slightly enlarged. The fluid was carefully sponged out, after which the abdominal parietes, the liver, and the intestines were well rubbed with a sponge in order to excite adhesions; the abdominal wound was then completely closed.

The lad made a good recovery after the operation; at first there was a slight re-collection of fluid, but this subsided without further tapping; the general anasarca, however, was considerable and lasted some months, but it has finally disappeared, and Dr. Passmore writes on April 9 that "the boy still has some albuminuria; but for this he is well, his parents take no troable to diet him, so he lives, eats, and takes exercise just as other boys."

Case 3. *Ascites and general anasarca due to cardiac disease.*—A little girl, æt. 9, was admitted into the Blackheath Cottage Hospital on August 2, 1900, under the care of Dr. Miller. She had enjoyed good health up to six months before, when she began to lose flesh and became tired after the slightest exertion, and for about two months she had complained of pain in her side, loss of appetite and of sleep; she had been treated as a case of tuberculous peritonitis. On

admission there was a considerable amount of fluid within the abdomen and very great œdema of the lower part of the abdominal wall, the legs, and thighs; the child was nearly always in a kneeling position, and never lay down. There was a loud systolic cardiac bruit and the liver dulness was increased; there was no albuminuria, and the amount of urine was not diminished (about twenty-six to thirty ounces being passed daily). She was treated by digitalis, etc., for three weeks, but as the œdema became more marked, incisions were made into the abdominal walls, allowing a quantity of serous fluid to drain away. The œdema and ascites, however, did not decrease, and as the child appeared to be going down hill, I was asked to see her on September 12. The abdomen was opened in the middle line above the umbilicus, and about eight pints of clear fluid were evacuated. There was no evidence of any tuberculous peritonitis, but the liver was found to be greatly enlarged and somewhat irregular in outline; the liver, together with the parietal peritonæum and intestines, was scrubbed with gauze to excite adhesions. The child rallied well from the operation, the œdema of the thighs and of the abdominal wall completely disappeared, and there was not any re-collection of ascitic fluid. The patient was able to lie in bed comfortably, and took her food well. The wound healed within ten days, and she returned home on October 4, able to walk about and without any ascites or dropsy.

On making inquiries I was disappointed to learn that the child died shortly after her return home from syncope, but there had been no return of the ascites. No autopsy was made.

Remarks.—Little need be said about the first case, except that it adds another to the list of cases of ascites due to cirrhosis which have been benefited by operation; although the patient died within six months from severe hæmatemesis following alcoholic excess, she was practically quite well in the interval, and there was no return of the ascites. The history of syphilis is interesting, as it is present in a considerable number of the other recorded successful cases, and it would therefore appear that the operation is more likely to prove successful when such a history is present.

The exact nature of the second case is still a mystery. I was inclined to consider that the ascites was dependent on some old tuberculous trouble, since one or two calcified

tuberculous deposits were found in the mesentery. It is hard, however, to explain all the symptoms by an attack of tuberculous peritonitis, since in the early stages the symptoms were those of acute nephritis, accompanied by attacks of abdominal pain. The intermittence of the albuminuria, and the complete recovery between the attacks, rather suggested that the albumen might be due to some pressure on the renal vessels or plexuses. At any rate, this case has been the most successful of the three, since he is still alive and in good health. The third case is interesting, as showing that this mode of treatment is also applicable to ascites due to cardiac disease. The child was in a desperate condition when I saw her, and not only was there no fresh collection of ascitic fluid, but the anasarca disappeared, and the child got up and was able to return home.

In looking over the literature on this subject, I find that a case somewhat similar to No. 2 has been reported by Talma, of Utrecht. This was the case of a lad, aged nine, who presented a mixture of nephritic lesions with enlargement of the liver and spleen, ascites, and general anasarca. He was operated on three times, the first operation being simply exploratory, which, however, was followed by rapid re-filling of the abdomen, gaping of the wound, and prolapse of omentum. The prolapsed omentum was cut off, and the wound re-sutured. During the following six weeks the abdomen required tapping four times, so a secondary laparotomy was performed, and the omentum was stitched to the abdominal wall; no further ascites occurred, but a third operation was undertaken in order to fix the enlarged spleen. A year later the child was in good health, the urine being normal, though the liver and spleen were still somewhat enlarged. This case was considered as a severe hæmorrhagic Bright's disease, with a hepatic complication which ended in an atrophic cirrhosis, with its usual complication of obstruction of the portal vein.

With regard to the technique of the operation, Morrison recommends that the omentum should be stitched to the abdominal wall, the intestines, liver, and spleen to be well scrubbed with gauze, and, after inserting a glass drainage-tube above the pubes, the abdomen is to be tightly compressed with strapping so as to favour the formation of adhesions; the drainage-tube is to be kept in for about a fortnight. Turner, on the other hand, advises that the liver and parietal peritoneum should be well scrubbed and then a piece of

omentum should be tucked in between the liver and the parietal peritoneum, and kept in position by sutures passing through all three parts; the abdominal wound is then to be closed without drainage.

The operation which I perform consists in opening the abdomen in the middle line above the umbilicus, turning the patient on one side to facilitate the rapid escape of the fluid, and then roughly sponging the intestines, and rubbing with gauze the liver and abdominal parietes; the liver is then stitched to the parietal peritoneum by silk sutures, without the intervention of any omentum, and finally the omentum is stitched to the parietal peritoneum in the region of the wound.

Recently Packard and Le Comte have recommended that, immediately after opening the abdomen above the umbilicus, another incision should be made just above the pubes; through this latter opening the ascitic fluid escapes by syphonage at the same time that the other steps of the operation are being completed. This certainly saves some valuable time.

The abolition of the drainage-tube I believe to be of great importance, since a great risk is run of the occurrence of septic peritonitis in a case where the large amount of serous discharge makes it very difficult to keep the dressing aseptic. A fatal result from this cause occurred in a patient operated on by Weir, and in another operated on by myself four years ago. It is therefore better to have some fresh collection of fluid, which can be drawn off again, than to run the risk of a septic peritonitis in a subject quite unfit to stand it. Since first writing this paper I have operated on another case of ascites from cirrhosis of the liver, which was accompanied by an irreducible umbilical hernia; it was noted before operation that the superficial veins round the umbilicus were greatly enlarged. After sponging out the fluid and scrubbing the contracted liver, a radical cure of the umbilical hernia was performed. The patient did well for a few days, but then developed broncho-pneumonia, from which she died on the sixth day. This patient was anæsthetised with ether, and in at least one other recorded case a fatal result was traced to broncho-pneumonia caused by the ether; in this case, however, I fear that the radical cure of the hernia may have contributed to the unsuccessful result by interfering with the collateral circulation which had already been established by adhesions of the omentum to the hernial sac. In comparison with this case it is interesting to learn that

Morrison's most successful case died two years after his operation as the result of a second operation for the radical cure of a ventral hernia.

With regard to the suitability of different cases for the operation, it is usually held that those in which the liver is still enlarged are most likely to be benefited, and that the operation will probably be of no use, and will often be followed by a fatal issue, when a small contracted liver is present. Renal and cardiac disease need not be considered as any bar to the operation, as is shown by Cases 2 and 3.

Although the operation has been followed by two deaths in my hands, I am rather inclined to think that these two cases of cirrhosis were not suitable for this method of treatment, and so I should not hesitate to recommend the operation in an early case of ascites from whatever cause, and if it is performed at this stage I do not think that there is any more risk than after a simple paracentesis.

The recorded cases of the operation up to date have been tabulated by Packard and Le Comte; including two of their own, they have collected twenty-two cases, of which five were fatal within fourteen days; three others died from the original disease between two and nine months after the operation, while three were not improved, two improved, and nine were cured. These authors, however, would exclude eight fatal cases on account of either inaccurate diagnosis or of faulty technique. Under these circumstances they state that the mortality of the operation varies from 7 to 23 per cent., while the percentage of cures varies from 40 to 64 per cent. Two other cases have been published since this table was drawn up, and adding these as well as my four cases we have a total of twenty-nine cases with the following results: eight died within fourteen days of the operation, five other cases died within nine months, four did not improve, two improved, and ten were cured. According to this, 35·7 per cent. of the cases are cured, and 27·5 per cent. died, the remainder being unaffected by the operation.

In considering the value of laparotomy in cases of ascites, I need not here refer to the cure of tuberculous peritonitis by this method, since this mode of treatment is now generally recognised and practised; I should like, however, to point out the good effect of laparotomy in cases of malignant disease of the peritoneum. Many have noticed the spontaneous diminution or even disappearance of growths after a simple exploratory operation, and in two cases of ascites dependent

on malignant in which I have deliberately opened the abdomen and scrubbed the peritoneum with a view of checking the ascites, the result has been so far satisfactory in that there was a considerable delay before tapping was again necessary, and, further, the subsequent intervals between the tappings was increased in length. This was specially marked in the case of a woman aged forty years, who was under the care of Dr. Finch-White in the Blackheath Cottage Hospital. She had required tapping at intervals of ten days before the operation, but did not require any further relief for five weeks after the operation, after which she only required tapping once more during the two months that she lived.

XXVI.—*Traumatic Subdural Hæmorrhage occasioning Convulsions on the Sixth Day after the Injury, and successfully treated by Operation.* By RAYMOND JOHNSON, B.S., and J. RISIEN RUSSELL, M.D. *Read May* 24, 1901.

FREDERIC W., a porter, æt. 33, was admitted into University College Hospital early in the morning of October 23, 1900, suffering from frequently recurring epileptiform convulsions, affecting chiefly the left side of the body.

The history, obtained from the man's wife, was as follows:—He was in his usual health until after a fall on the evening of October 17. He fell whilst carrying a sack of potatoes down a flight of stone steps, and his wife, who heard him fall, found him lying unconscious at the bottom of the steps, and bleeding slightly from a small scalp wound on the left side of the head. With the assistance of a neighbour he was got to bed, and remained unconscious till the morning—the combined effect probably of sleep, concussion, and alcohol; for his wife admitted that he was under the influence of drink at the time of the fall.

On the following morning he walked to see a doctor, who dressed the scalp wound, and prescribed some medicine, as he complained of headache and of being shaken by the fall. For five days the patient remained away from his work, but without apparently any particular symptoms, until after he had gone to bed at ten o'clock on the night of October 22, when he had a fit, and continued to have rapidly repeated fits until he was brought to the hospital early in the following morning.

On admission the patient was bathed in perspiration; the temperature was 102° F., and the pulse 100. Each fit lasted from two to four minutes, and the interval between two fits varied from one to five minutes.

Description of a fit.—The most usual mode of onset was either the turning of the head and eyes to the left, or twitching of the left side of the face; less frequently the movements began in the left hand, and still less frequently in both sides of the face simultaneously or even in the right side of the face. From the point of commencement the clonic

spasms rapidly involved the whole of the left side of the body and the right leg, and in some of the more severe fits the right arm also became involved in clonic spasms towards the end of the attack. During the fits the patient sweated profusely and was deeply cyanosed.

In the intervals the patient was usually sufficiently conscious to answer questions. The pupils were equal, of medium size, and reacted normally. No facial weakness could be detected, but the left arm was distinctly weaker and more flaccid than the right. The arm-jerks were present on the left side but not on the right. On the left side also there was exaggeration of the knee-jerk with ankle-clonus, and the extensor response was obtained as the plantar reflex.

With the object of controlling the convulsions, which had produced an extreme degree of exhaustion, chloroform was administered with success, and a full dose of bromide and chloral was given by the rectum. The chloroform inhalation was continued for about half an hour, but the fits quickly recurred when it was stopped, and the same result followed a second administration for a similar period.

In the consideration of the nature of the case two possibilities may here be mentioned. The first, which was not, however, seriously entertained, was that the convulsions were uræmic in origin, for the urine contained a dense cloud of albumen. The second possibility, which called for more careful consideration, was that the case was one of idiopathic epilepsy, brought out by a traumatism in an alcoholic subject with a strong predisposition to the disease, for we learned that the patient's father had died of epilepsy, and that a brother was subject to epileptic fits. In this connection it may be noted that, although the fits almost invariably commenced on the left side, the first movement was commonly the turning of the head and eyes to that side,—a mode of commencement that is too common in idiopathic epilepsy to be regarded as of much value in the diagnosis of a gross lesion of the brain.

Many fits, however, began by twitching of the left side of the face, and others by movements of the left hand, and this fact, taken in association with the weakness of the left arm, which was observed between the fits, the exaggeration of the left knee-jerk, and the presence of the extensor response on the same side, was regarded as indicating the existence of a definite irritation of the right cerebral hemisphere. The evidence at our disposal did not seem to justify the diagnosis

of a focal lesion, in that the fits did not constantly begin in the same region, whilst all the other phenomena observed could be as well accounted for by a general disturbance of the right hemisphere as by a limited lesion.

In the consideration of the nature of the lesion it may be remarked that, had the onset of the symptoms followed rapidly upon the injury, no hesitation would have been felt in regarding them as the result of an extravasation of blood over the right hemisphere caused by the blow on the opposite side of the head, the position of which was indicated by a small superficial scalp wound near the left parietal eminence. It was possible to explain the late onset of the convulsions by regarding them as the result of a simple non-inflammatory œdema spreading around a contusion or hæmorrhagic focus in the cerebral substance; but the chief difficulty in accepting this explanation consisted in the absence of any sensory or motor phenomena during the six days preceding the onset of the symptoms. Notwithstanding, therefore, the absence of any satisfactory source of infection, we were forced to the conclusion that the lesion was probably inflammatory in nature—in fact, a meningitis. In view of our definite opinion as to the existence of some organic lesion of the right hemisphere, it was decided at once to expose the latter over the middle of the Rolandic area. Accordingly chloroform was again administered with the result previously obtained, that the convulsions were again arrested. On removing a disc of bone with an inch trephine, the dura mater was found to bulge slightly more than naturally, and to present a bluish tint as if from blood beneath it. On incising the dura a small quantity of thick dark blood escaped. The opening in the skull was enlarged upwards and downwards with bone forceps, and the incision in the dura enlarged in a crucial manner to the full extent of the bone opening. In all between two and three drachms of blood escaped. The surface of the brain, although on opening the dura it bulged slightly more than normally, presented in every other respect a perfectly natural appearance. The divided dura mater was turned into position, and the scalp incision sutured. None of the bone was replaced.

No fits occurred after the operation. On the following day there was very considerable weakness of the left side of the body, which in the case of the arm amounted almost to complete paralysis, and it was nearly a month before the arm had completely recovered its full muscular strength. The

extensor response on the left side was present for four days after the operation, and for six days the right pupil was larger than the left.

The temperature rose to 105° four hours after the operation, and for four days varied between 101·4° and 104°; it then rapidly fell, and after the sixth day was normal. During the first week the patient was extremely restless, and at times delirious, and was obviously suffering from a mild attack of delirium tremens. The symptoms gradually subsided under appropriate treatment, and from the end of the second week until he left the hospital on the thirty-fourth day the patient seemed in perfect health.

In conclusion we would repeat, that the two points which have induced us to place this case upon record are—first, the long interval which occurred between the head injury and the onset of the convulsions; and, secondly, the possibility that the case might have been mistaken for one of idiopathic epilepsy.

XXVII.—*A case of Extreme Dilatation of the Left Auricle of the Heart.* By ISAMBARD OWEN, M.D., and W. J. FENTON, M.D. *Read May* 24, 1901.

DR. OWEN reported—On the 10th of last November I saw R. W., a woman aged 40, just admitted to St. George's Hospital on account of urgent dyspnœa. She was greatly emaciated, with a pale face and ashy complexion. She was unable to lie down, and was breathing, with obvious effort, at the rate of forty-four respirations a minute, largely by the aid of the accessory muscles in the neck, the action of the diaphragm being suppressed. The pulse was 104, small and irregular, equal on both sides; the legs and buttocks were œdematous. Her illness was stated to be of many years' standing, and she said she had suffered from rheumatic fever in childhood.

On examination, a heaving cardiac impulse was felt over the præcordia and generally over the lower part of the front of the chest, but an apex beat was clearly visible and palpable. It was greatly displaced, occupying a situation unprecedented in my experience, in the eighth intercostal space and in the posterior axillary line.

A blowing systolic murmur was heard over the whole cardiac area, and indeed over the whole part of the chest, accompanied by a sharp second sound. At times a præsystolic murmur appeared also present. Both murmurs culminated at the apex beat.

The right side of the chest at the back was dull from the level of the sixth dorsal spine to the base, and below the level of the eighth spine vocal fremitus was almost absent. These signs extended into the flank at the same levels. In front the dulness merged into that of the heart.

The right apex was duller than the left. The cardiac dulness extended beyond the normal in the second and third left spaces, but the limits were not clearly defined. The abdomen was distended, and the liver extended to the level of the umbilicus. The temperature was normal and the urine free from albumen.

There being urgent need of relief to the breathing, and no apparent reason to doubt the presence of effusion into the right pleural cavity, I ordered paracentesis. A trocar and

cannula, the size of a goosequill, were inserted into the sixth right space in the mid-axillary line. The trocar seemed to enter a cavity and did not vibrate.

When the tap of the aspirator was opened pure blood flowed into the receiver. It was not venous, though unduly dark for arterial blood. I suspended the operation and made a microscopic examination of the blood. Finding nothing but blood structures present, I ordered the needle to be withdrawn and the wound sealed. The patient seemed none the worse for the proceeding, though in no degree relieved.

I had to leave town that afternoon and did not see the patient again till the 12th. In the interval she was under the care of Dr. Cyril Ogle. On the 12th I found her in much the same general state as on admission. I made a fresh and more complete examination.

The apex beat, the murmurs, the dulness, and loss of fremitus were as already described. In front, the second right space was duller than the left; the third duller still; all below the fourth rib quite dull.

While there was no pulsation over the cardiac area, except at the apex-beat, pulsation, synchronous with that of the heart, was felt over the right front of the chest, from the second space to the seventh vertically, and laterally from the mid-axillary line to within an inch of the sternum. A systolic thrill was felt in the right infra-mammary region. It culminated at a point in the sixth space, half an inch outside the nipple line. Here also the systolic murmur had a second culminating point.

On the 13th my senior colleague, Dr. Ewart, kindly saw the patient with me, but we arrived at no precise diagnosis.

The patient died early the following morning without fresh symptoms and apparently by gradual asphyxia. A *post-mortem* examination was made by Dr. Fenton.

Dr. Fenton reported:—

Necropsy.—The pericardial sac occupies the entire thoracic cavity below the level of the fourth ribs. It lies with its long axis transversely, and has an extreme measurement of 10¼ inches, extending from the right parietal pleura, to which it is loosely adherent from the spine to the anterior axillary line, to the pleura of the opposite side. There is no general pericarditis, but posteriorly the two layers are firmly adherent over the left auricle. About ten ounces of blood-stained serum are present in the cavity. The right auricle and right and left ventricles occupy their normal relative positions, the

auricle and its appendix showing considerable dilatation. The left auricle is enormously dilated, and extends, to the right of and on a level with its fellow, some 4½ inches, a groove due to the interauricular septum defining the limits of the two; its capacity is 30 ounces, its greatest diameter 6¼ inches, exclusive of the appendix, which is also greatly dilated. The wall is remarkably thin, and in places translucent, but to the naked eye healthy; microscopically no active morbid processes are found. The left auricle has been tapped at the extreme point of the adhesion through the right axillary line, and there is a zone of recent adhesion around the puncture.

The mitral orifice admits four fingers. The edges of the valves are somewhat thickened and the chordæ tendineæ somewhat shortened and thickened. The other valves are normal. The ventricles are moderately hypertrophied and dilated.

The lungs are small and confined to the upper part of the thoracic cavity. The left lung is free, the right has its lower lobe collapsed and strongly adherent to the pericardium; there are the appearances of a little general bronchitis, the pulmonary substance being otherwise healthy. The right pleural sac contains a few ounces of blood-stained serum. Except in the case of the lungs there is no evidence of compression of other thoracic structures.

In the abdomen the liver and diaphragm are greatly depressed. The viscera show the usual appearances following morbus cordis.

The following, as far as I have been able to collect it, was the previous history of the case:

The patient's father, a painter, suffered from gout; on the mother's side was a history of consumption. The patient had an attack of rheumatic fever at an age variously stated as ten, eleven, and sixteen, most often as eleven, and was laid up for several weeks. She had suffered at times from rheumatism since.

From 1882, when she was about twenty-two years old, she suffered constantly from palpitation and difficulty of breathing.

She was first admitted to St. George's Hospital on April 28, 1888 (No. 668), suffering from these symptoms and from cough and expectoration. The hospital registers show that she was then well nourished and free from dropsy. Rhonchi were audible over the chest. The action of the heart was very irregular, and the pulse intermittent. The apex-beat

was two inches below and half an inch to the left of the nipple. The dull area of the heart extended up to the third rib. There was a systolic thrill at the apex, and a loud blowing systolic murmur culminating at the apex and replacing the first sound. The murmur appeared to begin rather before the ventricular systole. No extension of dulness to the right of the sternum is noted. The case was diagnosed as one of mitral stenosis. The patient remained in till May 9, having an attack of tonsillitis from May 12 to 20. On her discharge she was fairly free from symptoms, suffering only from dyspnœa on exertion. The pulse was 62, full, but compressible, very irregular, intermitting about every five beats. The physical signs were unchanged.

She was next admitted on April 20, 1898, being then thirty-eight. She was under my own head-card, though, owing to absence from town, I saw her but during the latter part of her stay. I have no private notes of the case, but from the hospital register I find that when admitted she was complaining of cough, dyspnœa, and epigastric pain after food. She said she was subject to attacks of pain in the præcordia, shooting down the left arm, and occasionally preceded by pain in the right side of the chest. Hæmoptysis had occurred occasionally during the preceding two years, once to the amount of half a pint. She had been attending the Brompton Hospital during this time. Abortion had been induced in her last three pregnancies, owing to obstruction to breathing and cardiac symptoms produced by the gravid state.

Since February 5 she had been laid up with "influenza, bronchitis, and pleurisy of the right side." Since that date dyspnœa had increased, and the legs had swelled occasionally. The patient was thin and sallow, and unable to lie down. The apex-beat was now in the seventh space and mid-axillary line, the patient being in a semi-reclining position. A faint thrill was occasionally felt at the apex. A blowing systolic murmur, well heard all over the heart and in the left axilla, culminated at the apex. The second sound was reduplicated and impure at the apex; in the aortic area the first sound was impure and the second clear. All the cardiac sounds were weak. There was no rumbling or snapping sound over the usual cardiac area.

The expansion of the chest in respiration was equal on the two sides. There was marked pulsation of the lower ribs on the right side, with a systolic thrill best felt just below the

right nipple. Pulsation was seen at a point just above the right nipple in the fourth space. The front and flank of the right chest were dull up to the level of the fourth rib in front. Over this area was heard a loud rumbling systolic murmur, also heard, though more distantly, in the axilla and over the posterior base. Vocal fremitus was absent from the dull area, and breath-sounds deficient; vocal resonance little affected. The posterior base was resonant, but over it breath-sounds, vocal resonance, and vocal fremitus were deficient, and râles were heard. The systolic murmur was well heard at the inferior angle of the left scapula. The left side of the chest gave no abnormal pulmonary signs. The liver was pushed downwards to the level of the navel; there was no ascites, neither was there any œdema or albuminuria.

An aneurysm being at first suspected, the patient was placed, from April 26, on Tufnell's plan of treatment, and given iodide of potassium in scruple doses. The treatment was suspended on May 5, owing to an attack of fever, with pain in the right flank. The fever and pain subsided quickly after leeching. The patient was out of bed by May 17, and left on the 25th with her symptoms materially relieved, but no change, as far as is recorded, in the physical signs.

For two or three months before her final admission last November the abdomen had enlarged. Six weeks previously she had suffered from pain in the loins, and since that time from cough, expectoration, and increasing dyspnœa, with severe pain in the right side of the chest, aggravated by deep inspiration. She could not sleep without morphine, and sweated freely.

At present I have little comment to make on this curious case. The observations of 1888 and 1898 show that the dilatation of the heart was progressive, and that it had not attained any unusual degree at the age of twenty-eight. The early age at which it apparently commenced, and the history of rheumatic fever in childhood, suggest, in the absence of any other apparent cause, that the primary cause of the dilatation was the adhesion of the pericardium over the left auricle. The dilatation of the mitral orifice was probably secondary to that of the cavities; the contraction of the valves themselves is but trifling. The persistent presence of a præsystolic murmur, slightly marked as it was, suggests that owing to the contraction of the chordæ tendineæ there was a hindrance to the onward passage of blood from

the auricle to the ventricle, amounting to a relative or absolute stenosis during diastole of the ventricle. The thickening and contraction of the cords and edges of the valves may have been due to the original rheumatic fever, but there is no other evidence of endocarditis.

The main difficulty in diagnosis lay in one's ignorance of the possibility of such a condition existing. The possibility once revealed, the significance of the physical signs is not difficult to discern.

The inaction of the greater part of the wall of the right ventricle, or the very slight action of the whole, whichever was the case, may account for the fact that no vibration was communicated to the trocar when inserted into it, as well as for the apparent absence of injury to the patient from the puncture. The fact of the puncture falling within (though only just within) the area of pericardial adhesion probably had a further protective effect.

At present I know of no similar case. When some account of this is in print I propose to make inquiries as to the existence of similar specimens in other museums, and to submit the results, if any, of my inquiries to the Society.

I may add that, having ascertained that the patient had been under Dr. Sidney Martin's care at Brompton, I wrote to ask him if he recollected the case. Dr. Martin replied, "I remember the patient whose notes you enclose. I examined her on March 23, 1896, December 29, 1897, and March 8, 1899, and on all these occasions found only the signs of the greatly enlarged heart (*cor bovinum*) of mitral regurgitation." In a subsequent communication Dr. Martin suggests, "perhaps some infective disease intervened to dilate the heart greatly." Of this the only evidence in the history is the doubtful account of "influenza" in February, 1898.

XXVIII.—*Acute Suppuration of the Thyroid Gland complicating Typhoid Fever.* By RICKMAN J. GODLEE, M.S. *Read May* 24, 1901.

ON November 29, 1900, I saw, with Dr. Christian, of Hounslow, a young lady æt. about 20, under the following circumstances. She took to her bed on October 27 with headache, pains in the limbs, a feeling of weariness, furred tongue, bleeding of the nose, and diarrhœa. Typhoid fever was diagnosed, and she was treated accordingly. The temperature, of which I append a chart, remained high—100° to 103° F.—till nearly the end of the third week in November. On the 22nd it was 98·4° to 100·6°.

On November 20 she noticed a swelling at the lower part of the neck, and next day there was a patchy, irritable rash all over the body, which disappeared on the 26th. By this time the swelling had much increased, especially on the left side, and the voice was thick and husky. On the 27th the swelling was larger, and caused pain up the side of the head and in the shoulder. These symptoms rapidly increased until the time I saw her, and in the meantime the temperature gradually rose, reaching 104·4° on the 28th, and 105·4° on the 29th, while the pulse, which had sunk to 92° on the 22nd, mounted to 140, and was very weak. She was at this time presumably in the fifth week of typhoid. She said that she had for some years noticed a swelling of the lower part of the neck, but that it was not enough to prevent her from wearing a low dress, and she took 13½ collars.

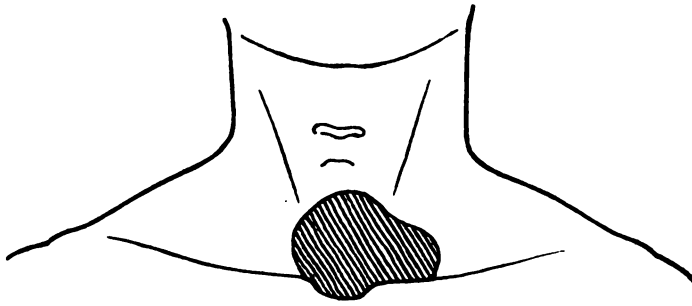
When I saw her she was evidently extremely ill, lying nearly flat, with an anxious expression, and perspiring freely; she had a weak and very rapid pulse, and some stridor which had come on during the preceding twenty-four hours. There was a marked swelling of indefinite outline as shown in the drawing (made from memory) in the lower part of the neck, occupying the middle line, and extending somewhat to the right, but much further to the left side. It was hard and obviously inflammatory, but gave a distinct sense of fluctuation both in the middle line and at the left side, though I could obtain no fluctuation from the central to the lateral part of the mass. The skin was not exactly adherent, but all the superficial

parts felt as if they were more or less fixed to the subjacent mass, and, in fact, like the surroundings of an abscess. There was no up-and-down movement on swallowing, though we could feel the larynx move during the act. There was some difficulty in swallowing.

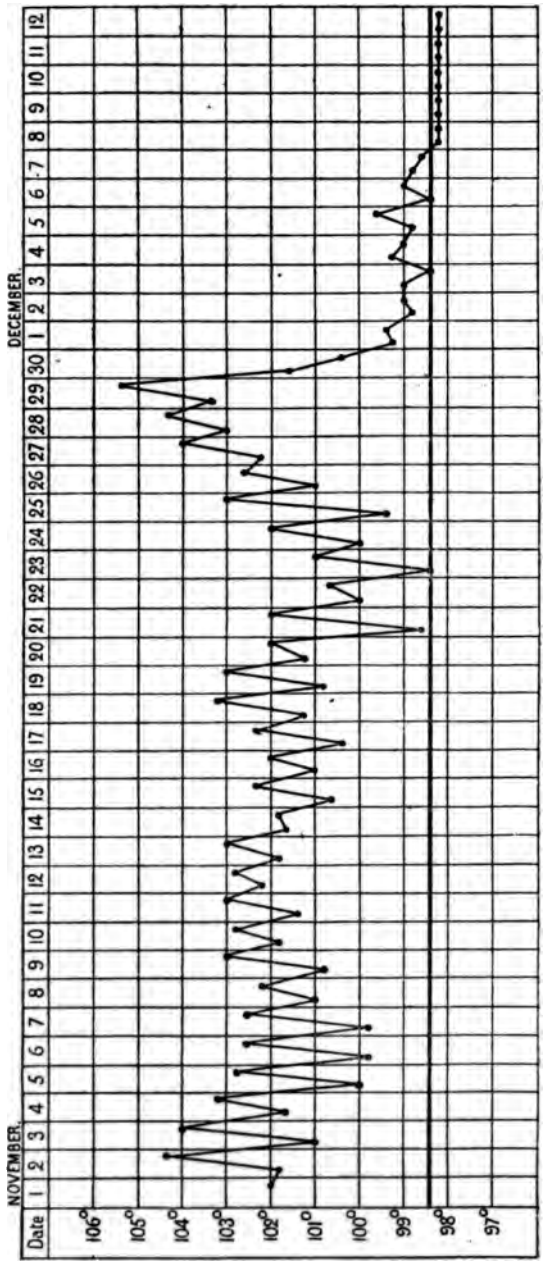
The eyes appeared to be prominent, but this was said to be normal with her. The pupils were large and equal, and reacted well to light.

There were no abnormal physical signs in the chest. I thought it was probably some suppuration superficial to the trachea and thyroid, and as matters seemed urgent, we at once gave an anæsthetic and operated by the very imperfect gas light.

Fig. 19.



An incision one and a half inches long was made in the middle line, which passed through inflamed tissues on to the much-enlarged thyroid without meeting with any pus. The gland was of a yellowish colour and a friable texture. I worked into its substance partly with the knife and partly with a blunt dissector, until a cavity was reached at a depth of about half an inch. The cavity was extensive, reaching to the extreme left limit of the tumour, and containing two or three ounces of fluid. The contents were principally a clear reddish-brown sticky fluid mixed with real pus in the deeper parts. The finger introduced into the cavity encountered masses of very soft material that felt like blood-clot, but were probably broken-down thyroid tissue. I did not pursue the search for fear of exciting hæmorrhage, but inserted a large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Date
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 1 2 3 4 5 6 7 8 9 10 11 12
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101°
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99°
98°
97°

drainage-tube three or four inches long, which immediately became filled with blood-clot. The bleeding, which throughout the operation was free, was not enough to cause anxiety. A cyanide gauze dressing was applied.

It was ascertained that the swelling had pressed the trachea considerably to the right; and when the patient recovered from the anæsthetic she expressed herself as comfortable, and it was observed that the voice had assumed its natural character.

Unfortunately I was unable to secure any of the pus for bacteriological examination, because it appears that in some similar cases the typhoid bacillus has been found as well as the micro-organisms of suppuration.

The progress of recovery was uneventful. The temperature fell after the operation to 101°, and rose during the night to 103°. It then rapidly fell to normal on the second day, and beyond two rises to 99·4° and 99·6° on the fifth and sixth days, remained normal afterwards. The pulse also quickly subsided to about 80. The discharge was very slight, and the tube was finally removed sixteen days after the operation, and now, March 12, she is reported to be in good health, and the swelling in the neck to have absolutely subsided.

There is a considerable literature with regard to acute inflammation of the thyroid in general and suppuration of the gland in particular. It appears that the acute inflammations may be divided into three classes:

1. Those which may be called idiopathic, in which the gland enlarges rapidly with more or less fever, and subsides spontaneously or after treatment.

2. The epidemic variety, which has been chiefly described by French military surgeons as occurring amongst soldiers, and which appears to be an affection of some gravity, and is clearly an acute specific fever.

3. The variety of which my case is an example. This occurs in the course of various septic diseases, such as the puerperal state, malaria, rheumatism, and, it is said, most, if not all, the acute specific fevers, especially typhoid fever.

I hesitate to refer to the bibliography of the subject, because I have not attempted to make an exhaustive search, and also because this has been thoroughly done by others, It may be stated, however, that without much trouble it was possible to collect accounts, some indeed not more than the mere mention of the case, of twenty-eight instances in which acute thyroiditis was found complicating the following con-

ditions. The table, to which I have added what it was possible to discover with regard to the proportion of cases which suppurate, must not be taken to indicate the relative frequency with which it occurs in connection with the several diseases enumerated, nor as a complete list of those diseases, for numerous others are referred to in the papers from which the records have been obtained.

Of these cases it will be seen that fifteen suppurated, and that in six the point is not definitely stated. Only one of the rheumatic cases is described as having suppurated, and as there was also a septic condition of the bladder, it was

	Cases.	Suppurated.	Did not suppurate.	Doubtful.
Rheumatism	8	1	3	4
Erythema nodosum	1	—	1	—
Puerperal conditions	4	4	—	—
Compound fracture	1	1	—	—
Malaria	3	1	1	1
Typhoid	2	2	—	—
Diphtheria	2	1	1	—
Hospital sore throat	1	—	1	—
Ozæna	1	—	—	1
Erysipelas	1	1	—	—
Pneumonia	1	1	—	—
Unascertained cause	2	2	—	—
Gastric disturbances	1	1	—	—
	28	15	7	6

probably a pyæmic case. In the well-known case of acute thyroiditis complicating erythema nodosum, described by Sir Thomas Barlow in our *Transactions*, vol. xxi, p. 67, 1888, suppuration did not occur. This seems to suggest that the rheumatic cases should, perhaps, be placed in a category by themselves.

The number of references to the occurrence of acute thyroiditis as a complication of typhoid makes me think that it is really more common than the foregoing table would lead one to suppose. It is, therefore, remarkable that I can find no reference to it in such text-books of general medicine as I have consulted, and that many physicians even of wide experience in this country appear to have met with no examples of it. It is for this reason that it has seemed right to draw attention to the subject by recording this solitary

case. It appears usually to occur in glands which were previously abnormal. In my case, it will be remembered, the patient had noticed a previous swelling of her neck. The inflammation usually goes on to suppuration, the character of the pus varying from the sort of material met with in this case to ordinary stinking pus. Numerous organisms have been found in it, amongst others the typhoid bacillus in cases complicating typhoid fever.

There can be no doubt that as soon as the signs of suppuration are well marked the proper course is to incise the abscess and drain it, and it does not appear that there is much danger of serious hæmorrhage. If treated in this way the prognosis is usually good, and recovery is likely to be rapid and complete. The plan of removal of the inflamed lobe, which has been practised by one or two Continental surgeons, is not, I think, likely to commend itself, as it must be a procedure of considerable gravity, and the experience of others, besides myself, tend to show that it is not necessary. At all events, it would be safer to defer it until the acute suppuration had subsided.

CLINICAL CASES

EXHIBITED.

*** Published in accordance with the regulation relating to the exhibition of patients at the meetings of the Society, viz. that each case shall be accompanied by a brief written description of the points it illustrates; such description to be published or not in the *Transactions* as the Council may direct.

I.—*A case showing the result of a Plastic Operation for the relief of Rectal Incontinence left after a modified Kraske's excision of the Rectum for Cancer.* By J. JACKSON CLARKE, M.B. *Exhibited October* 26, 1900.

JOHN F., æt. 57, was presented to the Society last session, together with specimen showing parts removed. The circumstances of the previous operation, already detailed, explained the presence of a gap extending from the anus in the middle line upwards for about two inches. The first operation was done on May 4, 1899. The patient was readmitted to the North-West London Hospital in June of this year in order that I might try to remedy the defect by a plastic operation, which, after due consideration, I had decided would be better for the patient than the use of a plug such as I had used in a former case of the same kind.

It will be found recorded in the account of the first operation how the lower part of the wound was necessarily left open, and how the lower one and a half inches, including the anus and sphincters, was laid open by a median proctotomy. Before the plastic operation was done there was a gap of

about two inches at the lower end of the anus in the middle line. A linear scar was all that remained to show where the sloughing of the lower end of the upper segment of bowel took place. The coccyx and the small last segment of the sacrum were removed at the first operation.

The plastic operation performed on June 14, 1900, consisted essentially of three parts.

1. Freshening the margin of the gap and dissecting up for about three quarters of an inch, all the bowel bordering on the gap.

2. Dissecting off the skin and fat for some distance on the left side of the gap, and on the right making a good flap of skin containing externally some fat and deep fascia over the gluteus maximus.

3. Suturing first the edges of the bowel from the upper limit of the gap to the edge of the skin, the part containing the external sphincter being made as broad as possible. Three silk stitches were passed, one above the other, through the middle of the flap; each of these stitches was passed first through the flap, then through the sacro-sciatic ligaments of the left side, back again through the same ligaments a little below the point of entry, and then back again through the skin flap one third of an inch below its point of entry into the same; then the suture was tied over a little roll of iodoform gauze. The skin edges were adjusted by silkworm-gut stitches. At the end of the operation a piece of drainage-tube wrapped in iodoform gauze was placed in the lower part of the rectum

The diagrams 20 to 24 may serve to render the description of the simple plastic operation more comprehensible. In fig. 20 the numeral "1" marks the fourth sacral vertebra, the fifth and the coccyx having been removed. "2" is placed just above the linear scar. The dotted lines indicate the manner in which the edge of the cleft was freshened. Opposite the scar "2" a piece of skin was included and turned in to compensate the narrowing due to the scar. At the ends of the sphincter a triangular surface was freshened. The interrupted line shows the extent to which the bowel was freed by careful subcutaneous work with a tenotome before passing the stitches, of which the upper three were passed like the upper one shown in fig. 20, *i.e.* tied with the knot inside the bowel; these were of chromicised catgut that had been kept for over fourteen days in $\frac{1}{20}$ carbolic; the lower was silkworm gut passed deeply and knotted outside. Fig. 21

FIG. 20.

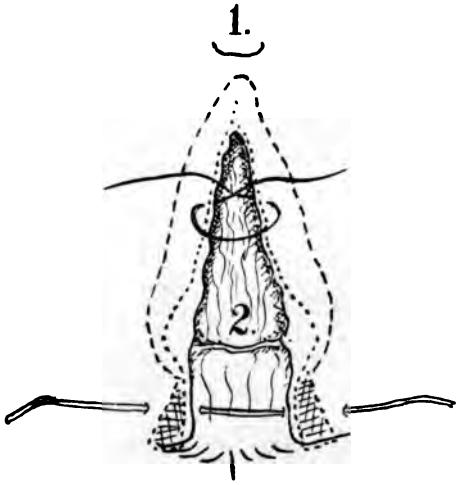


FIG. 21.

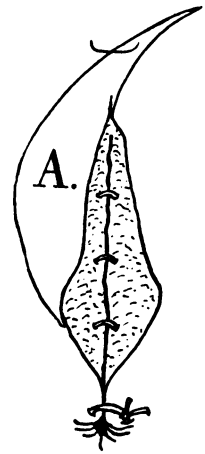


FIG. 22.

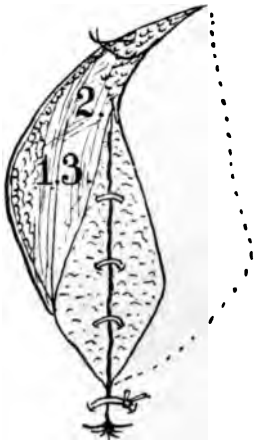


FIG. 23.

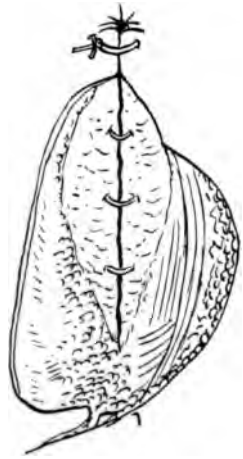
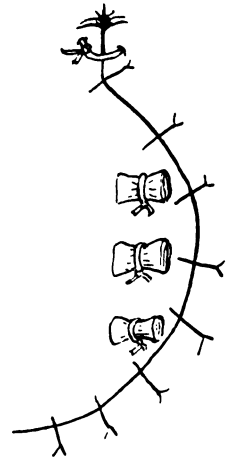


FIG. 24.



shows the effect of tying the sutures, and also two curved incisions that were made next in order to remove the skin and fat from the area "A." Fig. 22 shows the effect of the last-named proceeding and an interrupted line, which indicates the extent of undermining that was practised in making the flap on the right side. Some fibrous structures were exposed in removing the superficial tissues at "A," and were taken to be respectively, 1, the inner edge of the great sacro-sciatic ligament; 2, the fascia over the pyriformis, and 3 that over the levator ani. Fig. 23 shows the flap in the right raised and turned back. Fig. 24 shows the final stitching; the deep mattress-sutures are tied over rolls of iodoform gauze.

The healing was normal throughout. There was apparently no damage done to the nerve-supply of the sphincter. In a former case, where I had done a similar operation after the original method of Kraske, I left the lower segment of the bowel intact. A fistula formed. This I attempted to repair by a median plastic operation. In spite of my having done a preliminary colotomy the plastic operation failed. I subsequently read that this experience had not been uncommon. With these facts in view the need of a more secure way of closing the gap was evident.

The preparation of the patient had been such as the nature of the operation required. After the operation one quarter grain of morphia was given, and after this the bowels were kept quiet with laudanum, patient being on liquid diet till the 23rd; on this day the bowels acted comfortably with the aid of a dose of Epsom salt, and, subsequently, an olive oil enema. On June 30 Mr. H. O. Hobson, the Senior House Surgeon, records in his notes that the patient had no pain, was getting up, and had complete control over the bowel.

At the present time the patient is in good health. He has no sign of local recurrence or metastatic growth. The anus is almost normal looking, a small extension posteriorly being the only abnormal feature, but when the sphincter is voluntarily contracted this slight increase of aperture disappears, and the action of the muscle is very strong. The patient says that even under the stress of an attack of diarrhœa he has been able to hold back the contents of the bowel with perfect security.

II.—*A case of Myositis Ossificans Progressiva.* By J. WALTER CARR, M.D. *Exhibited October* 26, 1900.

WM. C., a boy æt. 7, first came under observation in May, 1900. He lives near Wimbledon; he does not appear to have had any previous illness except measles; his parents

FIG. 25.

are healthy; he has six brothers and sisters alive and well, and there is no evidence of rheumatism, syphilis, or tuberculosis in either his personal or family history.

Nothing abnormal was detected until about three months before he was first seen, when his mother noticed that his neck seemed stiff, and in a few days his shoulders, and then his elbows became stiff also. The history, however, is probably not very reliable. There is no evidence that the onset of his symptoms was preceded by any injury.

Present state.—The boy is fairly developed, but rather thin. His height is 3 feet 8 inches, and his weight about 42 lbs. He is of average intelligence for his age, and all his internal organs appear to be healthy. His temperature is always normal, and also the urine.

There is extensive but irregular ossification of the muscles of the back, particularly of the small muscles of the back of the neck, of the pectorals, latissimus dorsi, and biceps on each side, and of the left rectus abdominis; the tendons are especially involved. In most of these muscles, especially the biceps, the hardness is much more marked when they are put on the stretch. As a result of the affection of the above muscles the back is arched and almost rigid, both antero-posterior and lateral movements being limited; the head is held stiffly. Movements at both shoulder-joints are greatly restricted, so that the elbows can only be drawn about three inches from the sides of the chest. The elbows are fixed at a right angle, and cannot be extended beyond this, though they can be flexed through about twenty degrees. The chest is very rigid, and respiration mainly diaphragmatic. The leg muscles are not affected, except for a hard mass immediately below each great trochanter, and patient can walk well.

There is no alteration in the condition of the muscles when the boy is under chloroform.

Skiagraphy merely gives a slightly darker shadow than normal in the parts most involved, but nothing comparable with the shadow cast by the bones themselves.

Exostoses :—The following are present: one on each side of the forehead (one following a fall, the other a blow from a stone); on the distal end of the first phalanx of the second finger of the right hand; just below the inner side of the head of each tibia; on the left internal malleolus; and immediately beyond the metatarso-phalangeal joint of each great toe.

In addition, just below the spine of the left scapula, an isolated, flat, moveable piece of bone, over two inches long, can be felt in the subcutaneous tissue.

The disease has made very little progress whilst patient

has been under observation; some of the exostoses above mentioned have developed during this time, and the irregular bony prominences in the lower dorsal region have become more marked; on the other hand, the head can be rotated, and the arms abducted rather more freely than before. The boy does not seem to suffer any pain or discomfort.

As usually happens in this disease, there is a deformity, apparently congenital, of both great toes; they are turned outwards, *i.e.* towards the middle line of the foot, and are about half an inch shorter than the second toes, this shortening being due to synostosis and arrested development of the two phalanges in each toe. The thumbs, which are sometimes similarly affected, are in this case normal.

No special treatment has been adopted.

III.—*Radical Cure of a case of Chronic Frontal Sinus Empyema.* By Herbert Tilley, M.D., F.R.C.S. *Exhibited October* 26, 1900.

HANNAH C., æt. 50, complained for five years of a purulent discharge from the left nostril. During this time frontal headache was an occasional and troublesome symptom; frequently an unpleasant taste and smell were experienced; the throat was often dry, while each winter the voice was hoarse or very weak, "because of the dryness of the throat." A "winter cough" had during the same period been a source of annoyance.

During the patient's attendance as out-patient at the Golden Square Throat Hospital, the following facts were ascertained. The left nostril was obstructed by polypi bathed in yellow, non-fœtid pus. These were removed in the course of three attendances. The maxillary antrum was explored from the nose by Lichtwitz's trochar, and found to contain pus. This sinus was drained by the alveolar method, and irrigated twice daily with an antiseptic lotion (Boracic lotion gr. x ad ℥j). The anterior half of the middle turbinal was removed in order to remove obstruction from the lower end of the fronto-nasal canal.

May 15, 1900.—The external radical operation was performed. The anterior wall of the left frontal sinus was

exposed by a curved incision occupying the inner third of the eyebrow, and terminating just above the internal palpebral ligament. The bone was removed with chisel and mallet, and the polypoid degenerate mucous membrane lining the sinus curetted away. By means of specially curved burrs the fronto-nasal canal was enlarged sufficiently to admit the tip of the little finger passed upwards from the corresponding nostril. In doing this many diseased ethmoidal cells were also broken down.

A small drainage-tube was passed from above downwards, and its ends projected from the inner angle of the wound above and the nostril below. The sinus was packed with iodoform gauze and the wound sutured, except for the inner fourth of its length.

May 16.—Four inches of the gauze removed, and the nasal cavities syringed out with warm alkaline lotion.

May 17.—Gauze entirely removed, and sinus irrigated with warm boracic lotion. Nasal cavities syringed out three times daily from this time with a tepid alkaline antiseptic lotion.

May 22.—Similar treatment has been accorded, but rubber tube is now removed, and replaced by curved silver-wire plug.

May 29.—Sinus has been syringed out daily as before. The wire plug finally removed.

June 2.—External wound quite healed. Sinus syringed out from nose by means of special cannula. No pus. The antrum still continues to secrete a small quantity, although trans-illumination gives an equal light on both sides.

June 5.—Patient discharged, but ordered to syringe out antrum every day for three weeks.

November 18.—Patient quite well. No pus escaping from frontal sinus or maxillary antrum. The headache has been entirely relieved; there is no complaint of "dryness of the throat." The case illustrates the importance of—

1. Preliminary intra-nasal treatment, without which the external operation is futile and doomed to certain failure.

2. A free opening being made into the nose from the frontal sinus, not only in order to provide free drainage, but also to destroy the suppurating anterior ethmoidal cells around the fronto-nasal canal.

3. The importance of keeping the antrum free from pus, in order not to reinfect the higher sinus, as most often the antrum, ethmoidal cells, and frontal sinus are concurrently diseased.

The resulting scar in the eyebrow is scarcely noticeable, in spite of the fact that nearly all the front wall of the sinus has been removed.

[The case forms one of a series of fourteen, upon which a similar radical operation was performed; for fuller details of which the reader is referred to *Lancet*, July 14, 1900.]

IV.—*A case of Growth of the Left Tonsillar Region.* By StClair Thomson, M.D. *Exhibited October* 26, 1900.

THE patient is æt. 40. About the month of June last he accidentally noticed that there was something abnormal with the left side of his throat. It gave him no pain, and caused no symptoms.

It will be seen that the anterior pillar of the fauces on the left side is occupied by a new growth, which spreads upwards as far as the union of the soft to the hard palate, inwards to the margin of the anterior pillar, downwards to the side of the tongue, and outwards to the commissure between the upper and lower molar teeth. The margin is well defined and is not inflamed. The surface is flat and irregular, somewhat polypoid-looking, and bleeds readily when touched. It is not hard to the finger. The tonsil and posterior pillar on the same side are unaffected.

Although somewhat suspicious at first inspection, it will be noticed that there are no enlarged glands, no pain, and that the tongue is protruded easily, although the growth has affected its base. There is no history of specific infection, and the condition has been unaltered by the administration of mercury and iodide. The case has been under observation since the 20th of July, but the growth has not increased. On the contrary, the neighbourhood of portions which were removed for examination has not shown any tendency to ulcerate or proliferate.

Sections—which are placed under the microscope—show the structure of a papilloma.

P.S.—A deeper portion of the growth was removed later on, and under the microscope displayed the characters of a

squamous carcinoma. Although no enlarged glands were discernible, it was decided that the situation and extent of the growth rendered it inoperable.

V.—*A case of Intractable Ulceration of the Lips and Palate.* By G. R. Turner. *Exhibited October* 26, 1900.

C. DE LA L., æt. 58, joiner. For six months has had ulceration of lips; eighteen months ago ulceration was affecting gums, and all upper teeth were removed. Ulceration of lips became worse and very painful, preventing sleep at night.

Eighteen years ago had a chancre. No history of secondaries, etc. Has had a hernia for some years. Looks weak and ill. Has lost flesh lately. The lips are superficially ulcerated on nearly the whole of the inner aspect and edge, but very slightly on the outside. On the inner side there are many vesicles, with white sodden appearance, some pustules, and a few cracks. On the outside there are a few ulcers scabbed over, but many cracks and fissures running at right angles to line of lips. There is no redness around ulcers, and no induration, and the ulceration is very irregular in its distribution. Appetite good, but mouth is very sore. Tongue, thick white coat, rather tremulous. Nose and face unaffected. No enlarged glands felt.

On July 23 he was treated with mercurial powder locally and Pot. Iod. internally, and this was continued until September 13, but with practically no improvement. At a consultation it was thought to be a case of intractable ulceration of the lips, not malignant, or syphilitic, or tuberculous, and increasing doses of opium were recommended.

On September 28 arsenic was administered and the dose slowly increased. The ulceration is slightly better and the pain somewhat less as a result of treatment, but no marked change has occurred.

Dr. Rolleston, who on September 26 examined microscopically some scrapings from lips, reports:—"Necrotic mucous membrane with small-cell infiltration. No evidence of tubercle

DESCRIPTION OF PLATE X.

To illustrate Dr. Kidd's Case of Hæmophilia with Disease of the Knee-joint.

Bale and Danielsson, Ltd.

or syphilis." Has lately had local calomel fumigation with no good result.

It is to be noted that his son died of consumption. He attributed the ulceration of the lips, which was at first quite superficial, to the biting of a pencil. There was no tubercle bacillus discovered either in the sputum or discharges, and recently he has been injected with ·007 Koch's tuberculin without any result either local or general. Since he has been in hospital there has been a slight increase of new tissue about the margin of the growth on the lip, and an enlarged submaxillary gland has appeared on the left side. There is, however, no hardness either of the ulceration or gland.

VI.—*A case of Hæmophilia with Disease of the Knee-joint (with radiographs).* By PERCY KIDD, M.D. *Exhibited October* 26, 1900.

CECIL G., æt. 6. The patient, a fairly healthy-looking boy, a bleeder, and a member of a bleeder family, shows great swelling of his right knee-joint, mainly due to enlargement of the articular ends of the femur and tibia, as demonstrated by the accompanying radiographs.

The other bleeders in the family are one brother and a maternal uncle.

On admission (July 7, 1900) the joint was swollen, hot, and tender, and the skin over it was red and shiny. There was no definite fluctuation, but a vague crepitus could be recognised on manipulation. Numerous bruises were seen on various parts of the body.

During the first fortnight there was an irregular remittent pyrexia (103° to 99°), but since then the temperature has been normal. With rest in bed and splinting the pain and tenderness disappeared, and the swelling diminished. But on several occasions removal of the splint has been followed by renewed swelling of the joint, caused by the restlessness of the patient. The general condition has been excellent throughout, though bruises have frequently appeared as the result of trifling injuries.

History.—Five days before admission the boy fell on his

knee, and the next day the joint swelled and became painful. Two years previously he was treated in Poplar Hospital for arthritis of the knee-joint, which was regarded as tubercular. He subsequently made a good recovery, though, as his mother says, the knee always had a "funny shape." Profuse bleeding from slight cuts and injuries has occurred repeatedly from an early age, and on two occasions the boy was treated in the London Hospital and West Ham Hospital.

VII.—*A case of Multiple Cutaneous Carcinomatosis after Carcinoma Mammæ.* By H. D. Rolleston, M.D. *Exhibited October* 26, 1900.

A SINGLE woman, æt. 34, noticed a lump on the left breast three years ago. The breast was removed and the axilla cleared out on September 29, 1897, by Mr. Michels, who has kindly informed me that there was a very small growth in the outer lower part of the mamma, and that two small glands from the axilla were found to be carcinomatous.

There was no sign of recurrence till June, 1899, when a nodule was noticed under the skin over the *right* clavicle; since then the numerous subcutaneous tumours have gradually appeared. They vary from time to time both in size and in number. Many have grown larger, and have involved the skin, which is red and evidently infiltrated with growth. Those over the front of the abdomen have increased in size most, and the skin around them is in places becoming thickened, suggesting early "Cancer en Cuirasse." When she first came under my observation, in June, 1900, the tumours were all discrete, and felt like subcutaneous neuro-fibromata. Some of the growths, especially those in the right breast, have become more numerous. On the other hand, some of the tumours have undergone atrophy, notably one on the top of the scalp, while with equally careful counting the number seems to be diminishing. In June there were 105, and on October 22 78 discrete tumours.

The nodules are present in greatest number on the trunk; there are a few in the neck and on the scalp; there are also some enlarged glands in both axillæ and groins, and a few

nodules about the vulva and perinæum. Possibly the enlarged glands in the groin are secondary to nodules in the skin of the neighbourhood. A nodule was excised under

FIG. 26.

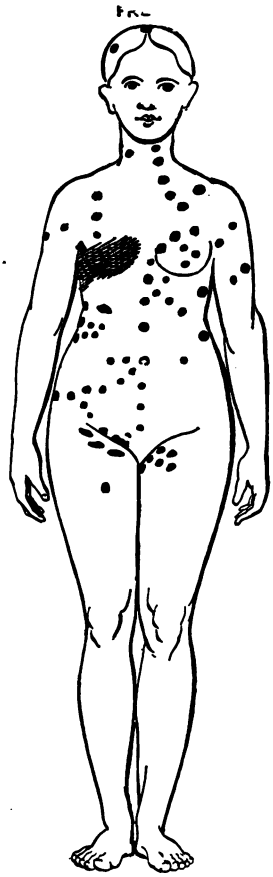
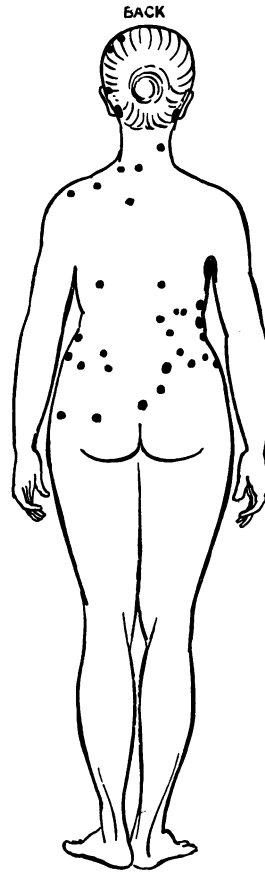


FIG. 27.



Diagrams showing distribution of the multiple secondary carcinomatous tumours on the skin. The left breast was previously removed for carcinoma. The shaded area over the right mammary region represents a number of closely-packed secondary growths. (105 tumours, not including mass in right mamma.)

Eucaine B, in September, 1900; a microscopic section, which is exhibited, shows spheroidal-celled carcinoma.

The condition is one of multiple cutaneous carcinomatosis due to blood infection, and not "Cancer en Cuirasse." It is further remarkable that in some instances the tumours are symmetrical, and that a large number of secondary growths have arisen on the opposite mamma, at all times a very rare situation for secondary growths.

The patient is very anæmic, grey-haired, thin, but not emaciated, and can hardly walk at all. From time to time she has severe attacks of vomiting; this dates from April, 1899, when she was obliged to give up her occupation as ladies' maid. The tumours are painful when they first appear, but not afterwards. There is no evidence that the liver or lungs are involved; in fact, in its manifestly cutaneous generalisation it resembles some cases of generalised melanotic growths following primary a melanotic tumour of the skin.

Treatment has consisted in the administration of arsenic and thyroid-gland substance. Removal of the ovaries was considered, but, inasmuch as it was thought this procedure would probably only modify the secondary growths on the right mamma, it was not advised.

The accompanying diagrams, drawn up by my late clinical clerk, A. T. Spanton, shows the distributions of tumours in June, 1900.

Note.—Since the above account was written, spontaneous fracture of the left femur near the neck has been reported.

VIII.—*A case of Cystic Kidneys, apparently congenital.* By Norman Dalton, M.D. *Exhibited October* 26, 1900.

THE patient is a woman, æt. 35, a general servant. She has had no illness of importance. She has had slight inguinal pains, and thinks that the abdomen has been enlarged for a year. It is more probable that she has noticed the enlargement because she has become thinner. The tumours

in the abdomen were discovered by her doctor when she was under treatment for dyspepsia.

Present condition.—There is a lobulated, elastic swelling on each side of the abdomen, occupying the position of the kidneys, but extending lower down. That on the left side is larger, and at times the colon can be distinctly made out in front of its lower portion. The urine varies in quantity, but is sometimes abundant. The sp. gr. is low (1012), the colour is usually pale, and sometimes there is a trace of albumen. As a rule she passes about half the normal quantity of urea per diem.

There has never been any dropsy, and there has been no uræmic symptom except occasional headache. On the other hand, there is marked loss of flesh and of strength. The pulse is tense and the heart apparently large, so that the case shows the cardio-vascular changes of Bright's disease. As there are no symptoms of stone, or any cause of hydronephrosis, the cystic condition of the kidneys is probably congenital. No alteration in the size of the kidneys has been observed since the patient was first seen in last June.

The condition of the heart deserves further notice. Its area of dulness is increased, and a remarkable wavy impulse from right to left can be observed. It appears a very large heart, and is probably larger than normal. But it is not so large as it seems, because there is a lordosis of the dorsal vertebræ, which has the effect of pushing the heart forwards and bringing more of its surface in contact with the chest wall. As regards murmurs, sometimes a systolic murmur has been heard all over the præcordium, but, more often, only a reduplication of the first sound at the place where the apex-beat should normally be, can be made out. In addition, a to-and-fro rub can be heard at the third left costo-sternal articulation. This cannot be due to recent pericarditis, as there are no symptoms of that affection. It is probably produced by a thickening of the pericardium (such as the white patches so often seen on the front of the heart), induced by its abnormal contact with the chest wall. The second sound of the heart is accentuated at the base.

IX.—*A case of Transfacial Bullet Wound.* By H. Batty Shaw, M.D. *Exhibited October* 26, 1900.

A. C., æt. 31, was a bombardier in the Royal Horse Artillery, engaged in South Africa; during a charge by the Boers at Diamond Hill, in about the middle of June last, the patient was shot with a bullet, which entered the face on the right side just in front of the ear; he was carried off the field unconscious, and remained so for eight or nine days. At the end of this time he noticed that his left eye was very much swollen, and discovered a hard mass just above the eyeball. This was explored, and proved to be a Mauser bullet. The bullet was removed, and the patient was soon able to be about. Various changes in his voice and difficulty in swallowing were noticed, however, and have persisted, though the patient says he is much better than he was, the paralysis being much less.

The case is exhibited on account of the number of nerves involved, and the peculiar course of the bullet. The following is a description of the various lesions found:

The sense of smell for substances like oil of cloves is greatly impaired in the left olfactory region, though not quite lost.

Acuity and the field of vision normal, and there are no ophthalmoscopic changes visible in the retinæ.

There is no paralysis of the third, fourth, or sixth nerves. Both pupils react briskly to accommodation, the left to light briskly, but the right only slightly.

No paralysis can be discovered in the muscles supplied by the motor division of the fifth nerve. Sensation in the face is deficient on the left side over an area extending from the left eye to the mouth, practically corresponding to the distribution of the infra-orbital branch of the superior maxillary nerve. In order to explain the paralysis of the right vocal cord it has been suggested that the right vagus may have been completely divided, and that the pulse rate was not increased, as happens in the experimental division of the vagus in animals. The mucous membrane of the upper lip and jaw is less sensitive on the left side than on the right.

The sense of taste is impaired on both sides of the tongue

for bitter, sweet, and acid impressions; no sensation of saltness appreciated on right side, but present on left.

There is some weakness of the left face, resulting in a little less definition of the left naso-labial fold. The electrical reactions of the seventh nerve are, however, quite normal on both sides. Hearing is quite normal. No lesion can be found particularly referable to the injury of the glosso-pharyngeal nerve, except some difficulty of swallowing.

There are no definite signs of lesion to the vagus proper, but the fibres which form the recurrent laryngeal nerve on the right side are presumably damaged, for the right vocal cord is reported by Mr. Raymond Johnson, who has examined the case for me, to be paralysed.

There is no lesion referable to the main trunk of the spinal-accessory nerve, but the palate on the right side is paralysed, and the patient suffers from regurgitation of liquid food and has a distinctly nasal voice.

The tongue is wasted on the right side, probably owing to division of the hypoglossal nerve. Though ordinarily the tongue on protrusion deviates to the right, yet the patient can thrust the tongue to the right or left angle of the mouth.

The sympathetic trunk has been apparently injured, for there are signs of paralysis of that nerve on the right side, as shown by ptosis and small pupil in the right eye. There is no exophthalmos in the right eye.

The patient suffers at present not only from the various paralyses indicated, but has pain behind and below the right ear. On pressing his fore-finger into the right auditory meatus he is at once seized with a desire to cough. The face has been radiographed, but no damage to the lower jaw on the right side was discoverable.

One cannot help but comment upon the patient's miraculous escape from a fatal issue. It is probable that all the present lesions are explainable by damage done to the various nerves outside the skull, but one is at a loss to explain why so peculiar a distribution of damage should have occurred near the angle of the right lower jaw.

X.—*A case of Hæmorrhage into the Spinal Cord.* By W. A. Marris, M.D. (for C. E. Beevor, M.D.). *Exhibited October* 26, 1900.

THE patient is a vigorous man, æt. 57, a road surveyor, much exposed to the weather. He has been a teetotaler for twenty years, and has had no venereal disease. His circulatory system is normal, but the urine shows a trace of albumen. In the early months of 1899 patient was slightly out of health. On April 24 of that year he suddenly felt a numbness in the left wrist, and then in the right. This passed away in a couple of hours, but returned next day while he was packing a small handbag. His hands at once became unable to hold anything; his left leg failed him, and he then felt a great "thump between the shoulders." He managed to walk downstairs, but half an hour later was unable to stand. There was no loss of consciousness. For some hours he had severe distension of the abdomen. At the end of a month, during which he had some difficulty in micturition, he was able to stand; but his hands were beginning to waste. His condition has remained unchanged except for some contractures.

At the present time he shows nothing abnormal in the cranial nerves, except that the left palpebral fissure is smaller than the right and the left pupil is smaller than the right, nor does it dilate so well to shade or under cocain (sympathetic paralysis). In the upper limbs the only change is found in the hands, where all the small muscles are atrophied, except a few fibres of the adductor pollicis in the right hand and of the abductor pollicis in the left. Both hands are thus in the "claw" position. In the left hand the fingers are more fixed in the flexed position than on the other side, owing to weakness in the long extensors, allowing of contracture by the long flexors. In the legs there is slight spasticity, more marked on the left side, but patient is able to walk at a fair pace without support. Sensory changes are present in the right leg and the trunk, but involve only his sensibility to pain and temperature, tactile sensibility being perfect everywhere. The loss of sensibility to pain and to temperature is found all over the right leg, the right half of the

body, front and back, up to a line one inch above the nipple anteriorly and to the spine of the scapula posteriorly. It is also found in a band round the left side of the trunk, between the levels of the umbilicus and the nipple in front, the last rib and the spine of the scapula behind.

The deep reflexes of the arms are equal, but more marked than normal. In the legs they are also increased, especially on the left side, where ankle clonus can be obtained. The plantor reflexes are both of extensor type. The sphincters are now acting naturally.

In addition to the wasting the hand muscles, and occasionally the forearm muscles, show a fibrillary tremor of a very coarse kind. The hand muscles have almost entirely lost their galvanic excitability, but those of the forearms all respond well to faradism.

XI.—*A case of Central Sarcoma of the Lower Jaw three and a half years after Enucleation.* By CHARTERS J. SYMONDS, M.S. *Exhibited October* 26, 1900.

EMILY D., æt. 16, was operated on on April 27, 1897. She had a central sarcoma in the left half of the lower jaw. There was an opening through the gum, through which the growth could be seen. The tumour was of large size, and bulged externally and internally. It was removed by an incision extending from the red margin of the lip under the jaw. The bony wall was divided and raised upwards as a flap. The lining membrane was completely enucleated from the cavity, the mucous membrane of the gum widely removed round the opening, the flap of bone pressed back into position, and the wound closed.

The special points exhibited were:—(1) freedom from recurrence after three and a half years; (2) the serviceable condition of the lower jaw; (3) the successful adaptation of the denture. The wrinkling of the skin is the only deformity exhibited.

XII.—*Oöphorectomy for Recurrent Mammary Cancer.* By STANLEY BOYD, B.S. *Exhibited January* 25, 1901.

F., ÆT. 31. Blow on the left breast three months before growth noticed. No family history; parents alive.

May, 1898.—Left breast removed by Mr. Davies-Colley.

January, 1900.—Operation for recurrence attempted by Mr. C. J. Symonds and abandoned.

August, 1900.—Very feeble and thin. Great difficulty in swallowing, especially fluids. Coughing is induced, and then vomiting of any food which has reached the stomach. Voice weak and cracked. Left vocal cord paralysed. Moderate amount of recurrence in scar fixed to chest wall; and one or two small subcutaneous nodules at a distance. Considerable enlargement of many left supra-clavicular and substerno-mastoid glands, the latter rather fixed; many large hard glands in right axilla, and a few in right neck.

August 8.—Double oöphorectomy.

August 15.—Improvement in swallowing, and voice rapid from day after operation. Skin wrinkling over larger nodules. Right axillary glands markedly smaller.

September 8.—Nodules and glands gone, except one beneath left sterno-mastoid. Voice still cracked.

December 4.—Voice and swallowing probably normal. Still something beneath left sterno-mastoid. 1½ stone gained.

Note (April, 1901).—Is apparently free from disease, and has gained three stone, not allowing for differences in clothing.

XIII.—*A case of Intractable Ulcer of the Forearm of undetermined origin.* By W. BRUCE CLARKE. *Exhibited January* 25, 1901.

F. W., æt. 20.

History.—Has had a lump on the back of her right forearm since she was ten years old, about the size of a walnut, painless; cause unknown.

In July, 1898, fell on some stones, and an ulcer formed.

October 6, 1898.—Became out-patient at St. Bartholomew's. The ulcer, which was at first about the size of the palm of one's hand, over middle of extensor surface of forearm, and looked like a broken-down gumma more than anything else, has gradually extended with indurated and everted edges.

August, 1900.—Whilst I was away the ulcer took on in parts a fungating character, and on my return to work in October, 1900, looked in parts like a possible epithelioma starting, and later on glands were felt in the axilla. These were removed in November, and submitted to microscopic examination with negative result. Pieces have been excised from the floor and sides of ulcer, and also with negative result.

The ulcer is now practically stationary, and no remedies appear to influence its progress.

XIV.—*Generalised Scleroderma with Raynaud's Disease.* By H. D. Rolleston, M.D., and S. Vere Pearson, M.D. *Exhibited January* 25, 1901.

A SINGLE woman, æt. 22, began to suffer from a combination of scleroderma and Raynaud's disease, which appear to have come on simultaneously six and a half years ago. The left ear has recently become slightly affected. The patient has never had ordinary chilblains, but in the last four years nine incisions for gatherings on the fingers have been necessary. She has been in the London, Charing Cross, and St. George's Hospitals; the constant current is the only treatment that has done any good; thyroid gland and nitroglycerine have been tried without benefit. She has had anæmia, gastric ulcer with hæmatemesis, appendicitis, and rheumatic fever, but with the exception of the last no joint symptoms. The catamenia are irregular and scanty. There is a family history of phthisis, but no evidence of hereditary or acquired syphilis.

There is a scleroderma of the whole of the face, suggesting, if the expression be admissible, double hemiatrophy. The skin is shiny, and so stiff that the eyes cannot be completely closed or the jaws sufficiently opened to allow the extraction of decayed teeth. The forearms and the lower parts of the arms are affected by scleroderma, which is advancing; supination and pronation of the arms are interfered with. There is slight scleroderma on both infra-mammary regions and on the right back and flank.

The fingers taper, are somewhat glossy, and are kept in a semi-flexed position, while movement is considerably restricted. They are usually tender and painful, being much worse in cold weather. There is some atrophy of the small muscles of the hands. The nails, especially over the index fingers, are curiously overcurved, but there is no clubbing of the fingers. On the tips of the right index and left ring fingers there is thickening from former ulcers. There are paroxysms of local syncope and asphyxia, the latter being much the more prolonged; occasionally the palms perspire very freely. The toes are also slightly affected.

The calves are very large when compared with the rest of the body. This enlargement came on four years ago after rheumatic fever. There have been attacks of pain in the legs, but no definite thrombosis of the veins. The knee-jerks are normal, but the skin over the calves is tender. The urine is healthy, and has never shown any abnormal pigmentation.

The association of scleroderma and Raynaud's disease, which has been recorded in about fourteen or more cases, suggests endarteritis obliterans as an underlying factor in both diseases.

XV.—*A case of Myasthenia Gravis.* By Risien Russell, M.D. *Exhibited January* 25, 1901.

W. C., male æt. 57. Family history unimportant. Good health until eleven months ago, when "blood-poisoning," owing to which right index finger was amputated. Has not been strong since, and his legs and right hand used to swell during the first six weeks after he left the hospital.

The weakness has been progressive for the past five months, at the beginning of which time his legs became rather suddenly weak one day, when walking, and he fell. After walking a short distance the legs begin to feel numb, and the knees give way, so that he falls; but on being assisted to rise he can resume walking. His eyelids drop at times, he sees double on looking to the left, speech becomes more and more difficult and indistinct during a conversation; he experiences difficulty in chewing and in swallowing, and liquids have occasionally regurgitated through the nose; he is also liable to attacks of dyspnœa and faintness, sometimes quite apart from any exertion.

The physical signs of his condition vary from time to time, and are always more pronounced after voluntary effort. The skin of the forehead is unduly wrinkled, and there is partial ptosis on both sides; but on looking upwards for a short time the muscles become fatigued, so that the wrinkles disappear and the ptosis increases. The pupils react to light and on accommodation, and it is not possible to fatigue the sphincter iridis by prolonged effort at accommodation, or by long exposure to a bright light, though the pupils oscillate under the latter condition. Slight nystagmoid jerkings are evoked when the eyes are first turned to either side, and convergence is defective. He is unable to close the eyes tightly, to pout the lips, or to whistle, and the upper lip is imperfectly raised when he attempts to show the teeth. The tongue is tremulous, but is not atrophied; he has difficulty in keeping it protruded, and all of its movements can readily be fatigued. The palate can be fatigued by making him vocalise the letter *a* repeatedly; moreover he experiences difficulty in speaking immediately after this, partly owing to embarrassment of respiration, his utterance becomes blurred and indistinct, and it has a nasal twang. Similar imperfections of speech can be induced by making him read aloud for a short time. He is unable to keep his arms raised above the head more than a few seconds before they begin to fall in spite of his efforts to prevent them. His grasp is feeble on both sides, and the fingers gradually relax their hold after a time, though fatigue is not so quickly or so completely induced as in the shoulder muscles. Expansion of the thorax is poor, and efforts at forced movements soon cause considerable embarrassment of respiration. The flexors of the hip and adductors of the thigh are weak and soon become fatigued, so that he has difficulty in crossing one lower extremity over the other when

sitting on a chair, and he is unable to keep the limbs in this position more than a minute or two before the upper one slips off. There are no spastic phenomena, and none of the muscles are atrophied. The myasthenic reaction has been determined in many of the muscles that have been subjected to an electrical examination. The knee-jerks are unduly active, and cannot be appreciably fatigued by repeated blows on the patellar tendon. Ankle-clonus is not present, and the plantar reflex is normal. The sphincters are intact, and there is no blunting of cutaneous sensibility.

XVI.—*A case of Elbow three years excised in a peculiar and unexpected manner for stiffness following fracture; unusually strong result.* By RUSHTON PARKER. *Exhibited January* 25, 1901.

R. P. D., æt. 30. Injured left elbow by a fall in India, November, 1896. Came to England with limb flexed to obtuse angle, and stiff; practically useless early in June, 1897. Excision performed July 5, 1897. Oblique fracture of outer condyle into joint, attached by soft material; severed with knife. Olecranon and head of radius cut off, and anterior ligament found very strong and likely to be useful, so not divided.

Inner condyle of humerus left, as it seemed likely to be a good lateral support to forearm bones, and likely to favour the hinge movement.

Result exceeded all expectations as to mobility and strength; enables him to ride and drive with freedom and confidence. The only movement defective is extension when arms abducted and prone, and that no detriment to him.

X-ray photographs, taken in December, 1900, accompany him. They do not clear up the case, strange to say, but rather confuse it, to my mind. The peculiar mode of dealing with the bones suggested itself in course of operation, which was previously intended to be a conventional excision. What was done was, under the circumstances, a great improvement, I think.

XVII.—*A case of Localised Muscular Rigidity.* By F. E. Batten, M.D. *Exhibited January* 25, 1901.

T. E., æt. 51. In 1889 had an attack of right hemiplegia, from which he gradually recovered under antisyphilitic treatment.

About the beginning of 1900 he woke up one morning and found his left shoulder swollen and painful, and at the same time he lost power in the left leg (slight left hemiplegia). In a few days the left leg was well again, but since that time the left arm has gradually become rigid.

The patient was first seen in September, 1900, and his condition was as follows:—The left shoulder-joint was firmly fixed, and all movement took place by rotation of the scapula. The arm was abducted and flexed at the elbow to a right angle. This, however, by forcible traction could be extended another forty-five degrees. This extension gave rise to very little pain. The hand could be moved freely. The muscles chiefly affected were the deltoid, supra- and infra-spinatus, triceps, pectoralis major, and biceps, and to a lesser degree the supinator longus.

There was no wasting, and the muscles were extremely hard and rigid, but not tender. Voluntary power in the muscles was still good, and passive movement gave rise to no pain.

Electrical reaction.—The affected muscles reacted only to a strong faradic current, the triceps hardly reacted, whilst to galvanism all the muscles reacted to strong currents, the K.C.C. being greater than A.C.C.

Under an anæsthetic, however, a very much better reaction to the faradic current was obtained. A skiagram was taken, but no change could be detected either in the bones or the joint.

Under an anæsthetic only partial relaxation of the muscles could be obtained, and the impression gained was that the deformity was due to muscular rather than to joint or bone change.

This patient was shown at the Clinical Society as a case of "myositis." Subsequently, however, a portion of the deltoid muscle was removed, and on microscopical examination appeared to be normal. The rigidity persisted in spite of antisyphilitic treatment, and he left the hospital without any material improvement.

XVIII.—*A case of Aortic Regurgitation apparently due to rupture of a healthy valve.* By LAURISTON E. SHAW, M.D. *Exhibited January* 25, 1901.

GEORGE T., æt. 21, a cook on board a steamer, is attending the out-patient department at Guy's Hospital for aortic regurgitation.

The accident occurred in December, 1899. The only illness prior to that date was an abscess in the neck. He had never suffered from rheumatism, chorea, or scarlet fever. Before the accident he believed himself to be in perfect health. He had followed his employment for four and a half years, and had not been absent a day on account of illness. He had not suffered from breathlessness or palpitation of the heart.

In December, 1899, while at sea, he was lifting a heavy saucepan full of soup, when the ship gave a sudden lurch, and in trying to prevent the saucepan upsetting the patient put forth all his strength and immediately felt a severe pain in his chest and became faint and collapsed. He remained in his berth for two days till the steamer reached Hamburg, where he was admitted to hospital and continued as a patient for seven months. On his return to England he presented himself for treatment at Guy's on account of dyspnœa, palpitation, and occasional cardiac pain, and was found to be suffering from aortic regurgitation.

XIX.—*A case of Dextrocardia, probably congenital.* By A. M. GOSSAGE, M.D. *Exhibited January* 25, 1901.

A. S., æt. 46, a tailoress, complained of weakness and of palpitation on exertion. Ten years ago she had rheumatic fever, and was told by her doctor that her heart was on the wrong side. Five years ago she was laid up with pain in the right chest for a fortnight. No other previous illness.

On examination the heart was found to be in the right

thorax. The apex-beat was apparently in the fifth space internal to the right nipple. The superficial cardiac dulness extended up to the fourth costal cartilage and towards the left to mid-line of the sternum. Heart sounds normal. The liver dulness was in its normal position on the right side of the body, and the stomach seemed to be in the normal position on the left. The two sides of the thorax moved equally, and neither side was contracted. No abnormality found in the lungs by physical examination.

Congenital displacement of the heart to the right without transposition of the other viscera is decidedly rare. Vehsemeyer* in 1897 could only find twenty-seven clinical cases reported in medical literature, two of which were confirmed post mortem. I have found one additional case† and references to two more,‡ which with Vehsemeyer's own case brings the number up to thirty-one. Fourteen cases have been found for the first time at the post-mortem examination,‡ and in many of these there was much congenital deformity of the heart as well. The reasons for regarding the present case as probably congenital are the correspondence of the cardiac position on the right side to the normal position on the left, and the absence of any sufficient cause for the displacement to be found either on physical examination or in the history of the patient.

XX.—*A case of unusually great Dilatation of the Heart.* By J. Porter Parkinson. *Exhibited January* 25, 1901.

STEPHEN S., æt. 22, a photographer's assistant. Both father and mother have had rheumatic fever. The former died of heart disease, the latter is alive, with no marked evidence of morbus cordis.

The patient has always been delicate and unable to run on account of palpitation, etc. Had "growing pains" when twelve years old, later rheumatic fever, being in bed six months, and invalided for a year; after which he has had constant dyspnœa on exertion. Since then he has had two

* *Deut. Méd. Wochschrft.*, 1897, p. 180.

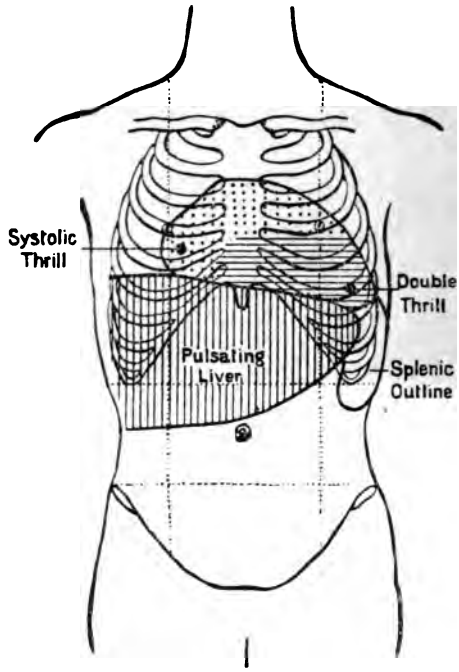
† Petit and Ravant, *Bull. et Mem. Soc. Méd. d'hôp. de Paris*, 1898.

‡ Berend, *Gyermetgyszgyaszat*, Budapest, 1898; Anclé and Bowyer, *J. de méd. de Bordeaux*, 1897, xxvii, p. 413.

other attacks of rheumatic fever, and for the last two months has had swelling of the feet and occasional vomiting.

When admitted to the hospital in November, 1900, he was pale and thin, slight jaundice, some blueness of lips and ears, slight clubbing, some œdema of feet, and dyspnœa on movement. Pulse 90, small in volume and irregular in size and rhythm. Jugular veins not very prominent, but the right

FIG. 28.



jugular sinus pulsates systolically. Much precordial bulging with systolic recession. H.A.B. Fifth intercostal space almost mid-axillary, line weak and diffused. Pulsation well seen on right of sternum below nipple, and much epigastric pulsation. At apex systolic and diastolic thrill, and below right nipple a thrill occasionally pre-systolic and occasionally mid-diastolic. The precordial dulness extended upwards to the lower border of the second rib, to the right a finger's breadth beyond right nipple line, and to the left as far as the

mid-axilla. At the apex were a rough pre-systolic and a blowing systolic murmur, while the second sound was not heard. Just beneath the right nipple was heard a rough pre-systolic murmur and a short systolic one. At the base the sounds of the heart were not well heard, and all over a systolic murmur was heard. There was no evidence of ascites; the liver extended downwards to two finger's breadth below the level of the umbilicus, and pulsated forcibly with the systole of the heart. Spleen just felt on a deep inspiration. There was some congestion of the bases of the lungs. Urine twenty ounces daily, pale in colour. Sp. gr. 1020. A trace of albumen was present. The slight anasarca disappeared rapidly.

Since November the heart has somewhat reduced in size, but otherwise there is little change. The patient has never had any marked cardiac symptoms.

An interesting point is whether the tricuspid disease is not a congenital condition due to intra-uterine endocarditis; the comparative absence of distress seems to point to such a conclusion.

There is but little to append to the description of the case; there is evidently great enlargement of the heart, especially of the right auricle, and double disease of the mitral and tricuspid valves. The comparative absence of symptoms is remarkable; there is hardly any cyanosis and but little dyspnœa except with exertion; there is hardly any epigastric pulsation and no enlargement of the jugular veins, though the liver pulsation is well marked. Though the patient's chest is unusually deep, there is marked impairment of resonance at and below the level of the tenth dorsal spine, due no doubt to the enlargement of the heart.

XXI.—*A case of Combined Sclerosis of the Spinal Cord.* By G. Newton Pitt, M.D. *Exhibited January* 25, 1901.

WM. H., æt. 32. He had a chancre followed by severe secondary eruption in 1894, and while under treatment an internal strabismus of the right eye developed. He emaciated and was ill for many months. The sight of the right

eye failed gradually, and has not improved since; the sight of the left eye he believes was not then affected.

He worked as a cooper until eighteen months ago, drinking enormous quantities of spirits, but this year he has usually had about half a gallon of four ale daily.

Four months ago noticed numbness in the left leg, which has increased, and now both are anæsthetic.

Three months' ataxic gait, worse at night. His gait now is both ataxic and spastic; he often stumbles and falls.

He has been unable to work as a carman since September, as the reins often fell out of his hands, and he did not know where they were. The anæsthesia has spread up the forearms, but is now less complete in the palms of the hands than formerly.

He was admitted into the hospital in December, under Dr. Hale White, but insisted on going out after three days; but the same day, after drinking two and a half pints of beer, he became unconscious, was readmitted, and remained in a state of stupor for two days. At first he was unable to hold a knife or fork, or feed himself, but now can manage if food is cut up.

Present condition.—He has complete anæsthesia and analgesia up to the knees and elbows, with the exception of some slight appreciation of touch on the palm of the hands. Complete loss of muscle sense. He is extremely ataxic both in hands and legs; no wasting. Movements slow and somewhat feeble. Intelligence and speech normal. Internal strabismus of the right eye, the vision in which is reduced to perception of light. The sight of the left eye has failed during the past few months, and began to fail two years ago. Both optic discs are hyperæmic, the margins indistinct and swollen, veins large, but no vessels are obscured, nor are there any hæmorrhages. The vision of the left eye is J. 18. Pupils equal and reactions normal. Movements of the left eyeball normal, except for a slight oscillation on looking to left. Headaches more or less frequently for the past four months, vertical and frontal. Sometimes he has cutaneous tenderness over the scalp. For three months he has had sickness three times a week, but only once while in the hospital. He cannot stand with his eyes shut. Knee-jerks exaggerated with ankle-clonus; no plantar reflexes. No exaggeration of the wrist-jerks. No anæmia, no pyrexia, no shooting pains. The movements of the right hand are especially stiff and defective. The condition, while it resembles the condition

described as subacute combined spinal sclerosis, presents many differences.

Additional Note.—It has since appeared that the man has been in many hospitals, and much of his history was wilfully false. It is probable that he has some syphilitic meningo-myelitis, but much of the sensory defect may be fictitious.

XXII.—*A simple form of Electric Light and Heat Bath.* By F. C. Eve, M.D. *Exhibited January* 25, 1901.

THE apparatus is very portable, and consists of three ordinary electric lamps of 32-candle power fixed on a wooden base, and protected by a wire cage. It may be connected by a flexible wire with any domestic lamp-holder, without risk of injuring wires or blowing fuses.

The limb, apparatus, and a thermometer are all placed together inside a chamber formed of blankets thrown over an ordinary cradle. The temperature of the chamber rises in fifteen to thirty minutes to 200°—250° F., and the bath is continued for thirty to sixty minutes.

The limb sweats copiously, and acquires a mottled erythema.

Pain invariably disappears, and the mobility of stiff joints is much increased. The patient sweats all over, and the body temperature is often raised to 101° F.

If the cradle employed is large enough to contain the whole body (except the head), it is necessary to employ two apparatuses to raise the temperature to 200°—250° F.

The treatment has been applied, so far, to eight cases of chronic joint disease, with the result that it has invariably relieved the pain entirely, and has considerably increased the mobility. In one case it produced freckling of the skin.

The advantages of the apparatus are that it is inexpensive, very portable, makes no smell, costs only 1½*d.* an hour in current, and it can be applied in any bedroom where there is an electric lamp, without moving the patient out of his bed or house, and exposing him to the risk of chill. The treatment can be extended over very long periods with trivial expense.

XXIII.—*A case of Multiple Fibro-neuromata.* By C. Beevor, M.D. *Exhibited January* 25, 1901.

CAROLINE S., æt. 9.

Family history.—Father had paralytic stroke seven years ago. Four other children died—one two hours after birth.

Previous.—Patient born with difficulty, and had paralysis of right brachial plexus at birth, involving especially deltoid, supra-, infra-spinatus, biceps, supinator longus, and other muscles, and from this she has recovered considerably. Has had slight ptosis of right eye and slight external strabismus since birth.

History.—About three months ago it was noticed that she had numerous "lumps" appearing on various parts of the body.

Present condition.—There are numerous small tumours scattered over the body. They are in the main subcutaneous, and the skin can be moved freely over them. They are small and in chains like beads on a string. For the most part they lie in the course of nerve-trunks, and are tender when handled. The chief places are—

Along the right and left supra-orbital nerves.

Along the left auriculo-temporal nerve.

Along the occipital nerves.

Along the nerves of both cervical plexuses, extending along the posterior triangles of the neck and over the clavicles.

Along the back on either side of the spine corresponding to the external branches of the posterior division of the dorsal nerves.

Along the course of the median nerve, and there is one large swelling in front of the internal condyle of humerus.

Along the lateral cutaneous branches of the intercostal nerves there are a few enlargements.

Along the anterior crural nerves slight enlargements.

This case differs from true Recklinghausen's disease in that there are no tumours of the skin such as molluscum fibrosum, and no definite pigmentation; but there are one or two spots, one over the inner end of the left clavicle and the other over the right scapular spine, which are of a reddish-

brown colour. In the latter place there is the impression of a pitting and as if the skin was thinned at this spot, and it is probably a trophic change.

Examination of an Excised Tumour by Dr. James Collier.

The tumour measured 15 mm. by 5 mm., and was of a regular elongated oval shape. Either end was continued into a small nerve.

The outer surface was smooth and shining, except at one spot, where it was adherent apparently to the deep fascia. On section it was hard, of cartilaginous (semitransparent bluish-white) appearance, and it was homogeneous. The tumour was split longitudinally, and sections were prepared by Van Gieson's, Pal's, Stroebe's, and by the Orcein method; one half of the tumour being prepared by the paraffin method and one half by the celloidin method.

Surrounding the mass was a dense thick connective-tissue capsule, in which were embedded two nerve-strands, each containing about one hundred nerve-fibres. A series of sections showed that one of these nerve-strands turned into the substance of the tumour, and its fibres became scattered through the substance of the tumour to re-collect after having traversed its length. The other nerve-strand ran from end to end of the tumour without leaving the capsule.

The substance of the tumour consisted of dense, white fibrous tissue arranged in irregular wavy bundles. At either pole of the spindle-shaped tumour this ground tissue was very dense, and had almost the appearance of closely set, non-striped muscle. Throughout the tumour widely separated nerve-fibres were visible.

XXIV.—*A case of Symmetrical Tumours of the Legs.* By J. Stewart Richards, M.D. *Exhibited January 25, 1901.*

H. W., æt. 30, was admitted to the St. Saviour's Union Infirmary on December 17, 1900, for otitis media.

He was found to have symmetrical tumours of the legs of six years' duration. The following history was elicited:—

Of early illnesses he remembers only earache. He has never had rheumatism. He has served twelve years in the army as a musician, 8¼ of which were spent in India and Ceylon. He left the army in 1897. In 1889 he had a chancre and a urethral discharge followed by a bubo, which suppurated. He was in hospital five weeks, but does not remember having a sore throat or rash, and received no treatment on leaving the hospital.

In 1893 he had ague in Colombo, which led to "slow continued fever."

He was in hospital from March, 1893, to January 1, 1894.

After two months' convalescence in Kandy he rejoined his regiment and took part in a year's exercise in "route marching," viz. daily marches of nine to fifteen miles.

He felt "very done up" after each day's march, and had severe pains in his calves associated with restlessness and a constant desire "to be on the move." He first noticed the lumps in his legs after five or six months' marching. They appeared simultaneously, were soft at first, and subsequently hardened. They have persisted since. In 1896 he returned to England. The rest of the voyage relieved the pains in the legs, but they returned as soon as he recommenced his military duties.

After leaving the army he became a waterside labourer, and frequently hurt his legs against trollies and barrows at the wharves. After a day's work his legs swell and become painful. He usually spends his evenings indoors, resting.

On admission there was a symmetrical enlargement of the back of each leg, extending from the middle downwards to the point at which the tendon Achillis begins to narrow, where it terminated in a prominent rounded spur which gave to the leg, viewed in profile, a posterior curve somewhat resembling that of the upper of a riding boot.

Each tumour moved with the superficial muscles of the calf and appeared to surround and infiltrate the upper portion of the tendon Achillis, the lower portion of the soleus and to extend upwards along the tendons of the soleus and gastrocnemii until it shaded off gradually into the mass of the calf about the middle of the leg. The circumference of each leg at a level 4½ inches above the internal malleolus was 10½ inches. Each tumour measured roughly 5 to 6 inches in vertical extent, 2½ to 3 inches in breadth, and 1½ to 3 inches in thickness.

The tumours were firm and much harder than the sur-

rounding tissues, giving the impression of dense infiltration. Their consistence was uniform but for one elastic spot over the posterior surface of the tumour on the right leg.

The skin over them was very slightly reddened, more tightly stretched, and less moveable than normal. After rest there was no heat or œdema. There was no superficial tenderness, and the tumours could be handled freely without causing pain. There were several pigmented, slightly depressed scars on the legs, and a few above the knees, but the patient attributed them all to injuries received at his work.

The femoral glands on both sides were enlarged and indurated.

There were no scars or signs in other parts of the body suggestive of syphilis. No enlargement of liver or spleen was detectable. Over each ulna there was a small, moveable, subcutaneous nodule, apparently fibrous in character. Patient stated he had had them for some years.

After walking exercise patient gets pain in the tumours and increased swelling with œdema and redness of the skin covering them and a slight œdema of the ankles.

Since his admission the patient has been at rest in bed.

He has taken the following amounts of potassium iodide:

From December	20, 1900,	to January	7, 1901,	15	grains	daily.		
„ January	7, 1901,	„	12,	„	30	„	„	
„ „	12, „	„	25,	„	45	„	„	

There has, however, been no appreciable diminution in the size of the tumours or alteration in their consistence.

Note.—May 10, 1901. After a prolonged trial of absolute rest, large doses of iodide of potassium, local inunction of mercury, and hot packs, the tumours remained unaltered.

XXV.—*A case of Chronic Lymphangitis.* By A. E. Bridger, M.D. (introduced by C. S. de Segundo, M.D.). *Exhibited January* 25, 1901.

M. M. I., æt. 58. For past thirty years has lived in Australia and subtropical climates.

Occupation.—Traveller.

Habits.—Always strictly temperate in alcohol. No venereal disease at all.

Disorders prior to the present one.—Occasional attacks of gout.

History of present disorder.—In June, 1898, both knee-joints greatly enlarged and very painful. Moderate fever. A doctor in Sydney tapped *the left knee-joint,* withdrawing probably (for 15 to 20 oz. were drawn off) all synovial fluid from that joint. The right knee-joint was not tapped.

Barely three weeks later patient attacked with pneumonia; the symptoms pointed to a septic form.

The excess of fluid in the right knee-joint was rapidly absorbed, and that joint recovered completely; but the left tapped knee-joint remained fixed, and œdema of the leg with great pain, described as of a rheumatic character, set in.

That was the state of matters two years and four months ago, and no improvement, in spite of excellent treatment in the colonies and of thirteen months' special treatment at the hot springs in Northern New Zealand, has since taken place.

The advice of two eminent surgeons and of one well-known physician was taken in August of last year, but no definite conclusion as to the nature of the disease, nor of any hopeful line of treatment, was obtained.

The patient was also taken to the Polyclinic, and was examined by Mr. Jonathan Hutchinson and some forty odd medical men, but with a like negative result.

Before that, at the end of August, Dr. Bridger at the suggestion of one of the surgeons consulted made a complete examination of the leg under chloroform. The skin was adherent to the deep fascia over a large area on the front of the thigh, the muscles were hard, the long bones of the leg were apparently thickened, the knee-joint was rigidly fixed, and the patella firmly attached to the bone.

At various times every drug likely to prove of service was tried in the colonies, and towards the end of last year Dr. Bridger, after a consultation, administered 1 grain of grey powder and $\frac{1}{6}$ of a grain of extract of opium thrice daily for three months, but without any improvement.

Hot soda baths, followed by thorough massage, have given some relief from the nocturnal pain—the cramps in the calf and thigh and the prurigo and eczema, which from time to time became almost unbearable.

The urine is normal, the bowels regular, the appetite fair, the strength maintained, and all the organs of the body are functionally and organically sound. An examination of the blood revealed nothing abnormal.

XXVI.—*A case of Cystic Hygroma of the Neck, probably congenital.* By StClair Thomson, M.D. *Exhibited February* 22, 1901.

THE patient, a woman æt. 29, presents a large irregular, soft swelling in the region of the upper half of the right sterno-mastoid. It is elastic and fluctuating, and is neither adherent nor inflamed. It causes no symptoms in the throat, and the patient complains only of its mechanical inconvenience and her fear of its getting larger, as she feels sure it has lately increased in size.

She is also positive that the swelling commenced just before her marriage (at the age of twenty-seven) as a small lump on the right side of the neck. This increased during pregnancy, and became larger after her first child was born in September, 1900.

It is stated that congenital serous cysts of the neck "are always noticed at or immediately after birth" (Bland-Sutton). Possibly, as they originate below the deep cervical fascia, the one in this case escaped notice until it had made its way through this membrane, and become superficial.

P.S.—The swelling was tapped with an exploratory needle, and some pale yellow fluid drawn off.

XXVII.—*A case of Extraordinary Curvature of the lower end of the Femur, following Epiphysitis.* By J. S. Richards, M.D., B.S. *Exhibited February* 22, 1901.

E. S., a female child, æt. 6 years.

In 1895 the child suffered from acute epiphysitis of the lower end of the right femur, involving the knee-joint. The joint was opened and treated by irrigation. The child was kept in a horizontal position until June, 1896, when she con-

tracted scarlet fever, and was sent to a fever hospital. She was readmitted November, 1896, fitted with a splint, and sent home.

In 1897 she was admitted to the St. Saviour's Union Infirmary. There was then a flail-joint and a slight curvature of the lower end of the femur. The child could throw the leg and foot across the shoulder of the same side.

In 1898 the child was returned to the hospital at which she was first operated upon, and the leg was amputated at the knee-joint. Soon afterwards she was sent for twelve months into the country.

On her return to the infirmary from the country the lower end of the femur presented an acute hook-like curve, with the condyles directed inwards with a decided twist forwards. The amount of the curve is well shown in the skiagram, though it gives but little idea of the direction.

The skin showed thickening and roughness over the point of greatest convexity, but there was no sign of pressure on the end of the stump. The child is rarely seen to place any weight on the roughened surface, and her favourite method of progression when not upon her crutches is to swing herself along supported by her outstretched leg and right arm, sliding the right buttock on the floor and keeping the stump flexed and adducted across the pubes.

Measurement along the convexity of the curve shows the right femur to be about the same length as the left, but that along the concavity is apparently much shorter. The curve, therefore, is in all probability due to inequality of growth.

XXVIII.—*A case of Progressive Myositis Ossificans.* By H. D. Rolleston, M.D. (for F. W. Burton Fanning, M.D.). *Exhibited February* 22, 1901.

MALE, æt. 33 years. Disease began at age of eight years. From the investigations of Dr. Vaughan, who is publishing the case *in extenso* in the *Lancet*, it appears that the father of the patient had the same disease. The patient is married, and has a daughter aged seven, who has not shown

DESCRIPTION OF PLATE XI.

To illustrate Dr. Richards's Case of Curvature of the lower end of the Femur.

Bale and Danielsson, Ltd.

any sign of the disease. No history of rheumatism. The disease is very advanced. All four limbs are almost fixed, thus resembling Marie's Rhizomelic spondylosis; the spine, except in the cervical region, is rigid from ossification of the muscles. Respiration is diaphragmatic, and patient cannot sit down; he can just walk. Movement of temporo-maxillary joint greatly limited. The thumbs are natural, but the great toes are markedly dwarfed (microdactyly). There is ankylosis of the terminal phalanges on the great toes and the fourth toe on the right foot. There are exostoses round the knee-joints, and one the size of a marble on the left side of the chest.

The ossification of the muscles is very widely spread.

XXIX.—*A case of Peculiar Affection of the Joints.* By Hugh Thursfield, M.D. (for A. E. Garrod, M.D.). *Exhibited February* 22, 1901.

W. B., a boy æt. 10, brought to the hospital for enlargement of right elbow and left knee, which has been present for four years.

Good health as a baby; fifth of eleven children, five other living children all healthy.

Occasional attacks of abdominal pain, which pass off quickly, and return only at long intervals.

No rheumatism or phthisis in family history; no history or evidence of syphilis.

The swellings of the joints began four years ago, and have steadily progressed.

The boy has congenital ptosis and the joint swellings, but is otherwise healthy and intelligent, though rather dull mentally.

? Nature of joint affection.

Report of Sub-committee appointed to consider Dr. Thursfield's Case.

We are of opinion that the patient has tuberculosis and some degree of hypothyroidism. One of us (J. J. C.) was

convinced that there was evidence of active rickets in the ribs and the left genu valgum.

WM. EWART.
J. JACKSON CLARKE.
April 15, 1901. H. D. ROLLESTON.

I agree that the patient has some degree of hypothyroidism, but do not think he has tuberculosis or active rickets. I think the condition in the joints more nearly resembles that seen in Charcot's disease, and some other affections of the nervous system.

THOS. H. KELLOCK.

April 22, 1901.

XXX.—*A case of Enlargement of the Right Foot; ? cause.* By H. BETHAM ROBINSON. *Exhibited February* 22, 1901.

MALE, æt. 38, a baker. Nineteen months ago had a definite injury to his great toe, followed by swelling, and the foot is said to have turned black. A few days after lumps were noticed on the right shoulder, on the outer side of the right wrist, and over the right knee. These went away by themselves at the expiration of a few days. This was succeeded by great loss of flesh, for which he was dieted, but he does not know if his urine was abnormal at the time. There was some suppuration about the toe-nail, which has continued slightly up to the present date.

He has now gigantism of the right foot, especially of great and fifth toes, and over tarsus. There is thickening of the phalanges and metatarsal bone of the great toe, with some looseness and crepitus in the joints, also thickening of fifth metatarsal. An X-ray photograph shows bony thickening about the phalanges and metatarsal bone of the first and over the same bones of the fifth toe. He has neuralgic pains about the leg, and on both sides his patellar reflexes are feeble. There is no difference in the size of the legs or arms, but some neuralgic pain down the left arm. The crests of the ilia seem a little thickened. There is a very marked bend in the lower cervical and upper dorsal regions of the spine. The head and face bones are normal. Pupils respond actively to light.

DESCRIPTION OF PLATE XII.

To illustrate Mr. Robinson's Case of Enlargement of the Right Foot.

FIG. 1.

FIG. 2

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The nature of the case seems doubtful. The question to be solved is, what relation the present condition bears to the injury? If not related, is the thickening of the bones only osteo-arthritic? in which case were the lumps on the arm, etc., rheumatic nodes, or is the enlargement of the foot acromegalic.

XXXI.—*A case of Actinomycosis in Parotid Region.* By Raymond Johnson. *Exhibited February* 22, 1901.

THE patient, a man æt. 31, a Russian by birth, who had been in this country for four years, came to University College Hospital on February 2, 1901, on account of a chronic inflammatory condition of the skin in the right parotid region, of eight months' duration. There is nothing to note in his history. He is now a tailor's presser, but when in Russia had a good deal to do with the buying and selling of horses. The affection began with pain and swelling of the posterior part of the right cheek.

Early in August, 1900, and again six weeks later, abscesses were opened below the right ear, and since that time slight discharge has frequently occurred from small openings in the skin. There is now a patch of indurated and congested skin over the right parotid gland, which also seems involved in the induration. The mouth cannot be freely opened, but the jaws and the cavity of the mouth appear healthy. At the time of the first examination there was a small sinus in the skin, from which a drop of pus was expressed, which contained yellow grains, presenting under the microscope the characteristic appearances of the ray fungus. The surface of the skin in the affected area is rendered slightly irregular by the healing of numerous small openings from which pus has from time to time been discharged.

It was subsequently learnt that the acute abscess, which was the earliest symptom, was in connection with the lower jaw.

After the patient was exhibited iodide of potassium was given in doses increased to 20 grains three times a day, with rapid and very marked improvement.

XXXII.—*A case of Acute Bronchocele.* By SIDNEY PHILLIPS, M.D. *Exhibited February* 22, 1901.

G. W., æt. 18, draper. Always good health, leads an active life, and was playing football till Christmas, 1900. Had a cold in January, but continued work. A small boil came on right side of face; he cannot give any cause for this; it was poulticed, burst, and soon healed, but he had pain and an enlarged gland behind the right ear, to which iodine was applied by medical advice on January 23. On January 25 noticed swelling about the thyroid upon the right side, which has extended to the left side. About the same time got breathless, and voice has altered during the last fourteen days.

Present state.—Very great enlargement of thyroid gland, both lobes and the isthmus, and a hard outlying nodule can be felt at the lower part of the left lobe; no thrill and no pulsation of vessels in the thyroid can be felt, but there is transmitted pulsation of the carotid artery, especially on the left side. Voice is altered; no cough. Breath short. Tongue always feels dry; no epistaxis. Temperature 100°. Pulse 90—160 per minute; slight systolic (? hæmic) murmur at the apex. He has not noticed that the eyes have become prominent.

March 18, 1901.—Since exhibited on February 22, 1901, the thyroid has remained about the same size; but a fine thrill has developed in the vessels on the right side of the neck, and throbbing of the thyroid gland itself has become marked; there is also evidence of paresis of the superior oblique muscle of the right eyeball.

XXXIII.—*A case of Tachycardia (? Graves's Disease).* By SIDNEY PHILLIPS, M.D. *Exhibited February* 22, 1901.

J. T., æt. 28, compositor, at the beginning of November felt tightness across chest, soon after legs and feet swelled; admitted to St. Mary's Hospital, December 24, 1900, with apex-beat too feeble to locate; pulse very feeble, 150; œdema of legs and such breathlessness that he could not walk many yards. Since then heart-beat has become stronger, with evidences of dilatation and hypertrophy; pulse rate has persisted at 140 to 160, with occasional temporary drops to 110 a minute, extremely feeble, and uninfluenced by digitalis or strophanthus or rest in bed. There is slight fulness of the right lobe of the thyroid; no albuminuria; no epistaxis; no tremor; no proptosis; occasional slight pyrexia.

J. T. had not altered in any way by March 18; the heart rate persists at 140 to 150 per minute, notwithstanding the administration of arsenic, of opium, and the use of Leiter's tubes.

XXXIV.—*A case of Injury to Left Optic Nerve.* By R. J. GODLEE, M.S. *Exhibited February* 22, 1901.

INJURY to the left side of the head from a fall from a bicycle, followed by paresis of right side of face and right upper and lower extremities, deafness of left ear, and blindness of left eye. The paresis of face and limbs has much improved. The deafness is much less, and probably depended upon injury to tympanum. The blindness continues. There is evidence of injury to the left optic nerve, *i. e.* optic atrophy, and a pupil not reacting to light on direct stimulation, but acting briskly when the stimulation is applied to the right eye.

XXXV.—*A case of Congenital Morbus Cordis. ? Patent Ductus Arteriosus Regurgitation.* By PERCY KIDD, M.D. *Exhibited February* 22, 1901.

SUSAN G—, æt. 21, cigar maker. Cyanosis slight, especially of conjunctiva; no clubbing.

Heart enlarged, right side especially. Faint diastolic thrill in second left space. Second sound everywhere very loud; maximum intensity at third left cartilage. At this point a short diastolic murmur following second sound audible at times. First sound everywhere feeble, and generally doubled.

History.—Attacks of cardiac pain from childhood. Dyspnœa for three years. No rheumatism, scarlet fever, or chorea.

XXXVI.—*A case of Bullet Wound of the Brain, showing injuries to the Optic Nerve and the Pyramidal Tract, corresponding to the course and present position of the bullet.* By W. G. SPENCER. *Exhibited February* 22, 1901.

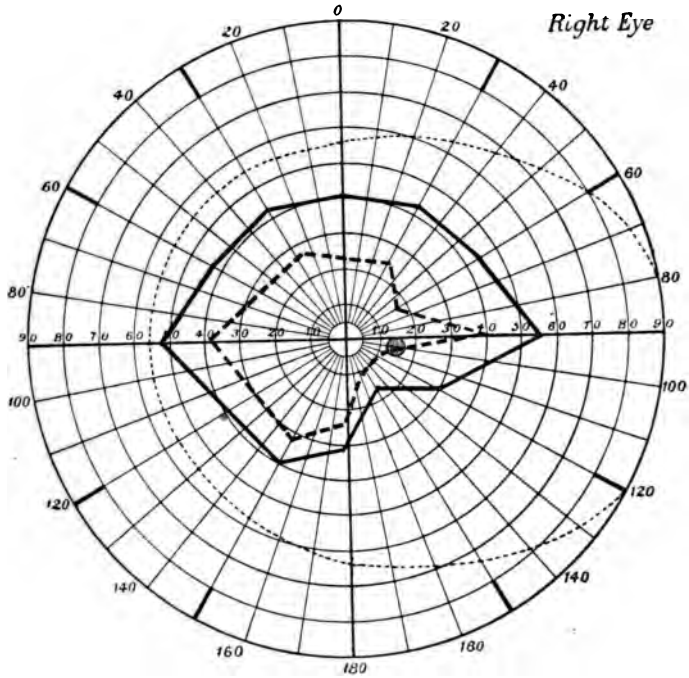
HISTORY.—A man now aged 25, in October, 1899, was wounded by a pistol bullet fired at close quarters from the right side of the bed on which he was then lying. The bullet entered immediately below the centre of the right lower eyelid and there remains a visible scar. He was taken to St. Bartholomew's Hospital, where he was under Mr. Walsham for two months. He was at first unconscious, with hæmorrhage into the left orbit, and later showed right hemiplegia and aphasia, with blindness in the left eye. Rigidity of the right arm with painful tremors began to be marked about a year ago. He has had no fits of any kind.

Present state.—He is in good health except for the results of the injury and a slight alveolar necrosis of the left lower jaw which requires surgical treatment.

Left eye: The pupil is contracted; left, 3·5 mm., right, 4 mm. There is no sympathetic reflex; it reacts well to light, both directly and indirectly, but slowly to accommodation. The movements of the eyeball are good except outwards, the action of the external rectus not carrying the eyeball outwards beyond the middle line. Vision is limited to perception of light; the disc is very white.

Right eye: The field of vision is diminished, and this is especially the case in the lower and outer quadrant (see peri-

PERIMETER CHART.



Dark continuous line, field for white. Dark broken line, field for red.

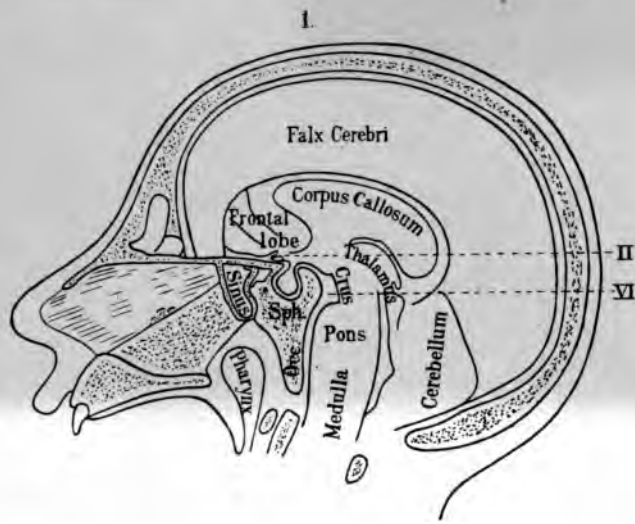
meter chart). The disc is not quite clear, but the vision is otherwise good.

Speech is now good except that he is sometimes at loss for a word.

Head: There are some fine nodding movements when he is disturbed.

Face: The right lower face is paretic without spasm. The upper face is normal, as also is the tongue. Sensation is normal, as also is taste and smell. He has no pain.

Right arm: Voluntary movements are absent, except a slight advancement at the shoulder. There is a constant



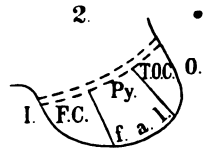


FIG. 29.—Diagram 1: A sagittal section through the skull and brain to illustrate Plate XIII.

II. Marks the optic chiasma and the origin of the optic nerve.

VI. The point behind the posterior clinoid process in front of the pons and crus cerebri where the sixth nerve enters the dura mater.

Diagram 2: A section of the crusta, showing the position of the fibres. I, inner side. O, outer side. F.C., frontal and cerebellar fibres. T. O. C., temporal, occipital, and cerebellar fibres. Py., pyramidal fibres. f, face. a, arm. l, leg.

tonic spasm with clonic movements superadded which become marked with any emotional disturbance. The tonic spasm keeps the arm in the position which can be imitated by putting the arm behind the back and opening the hand with the palm

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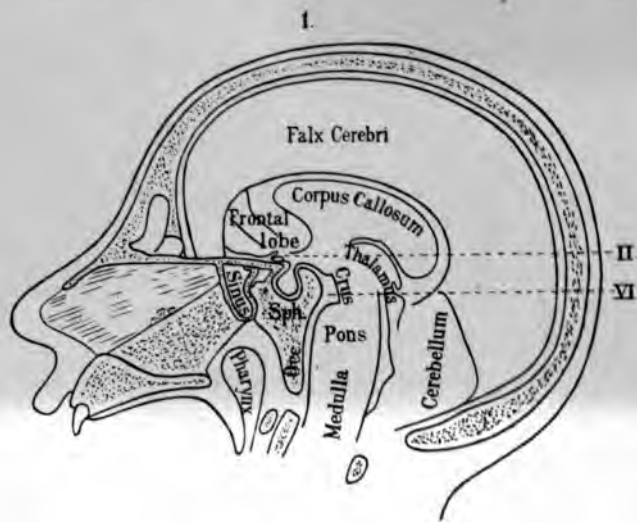

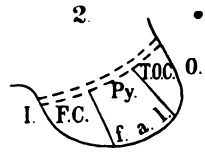

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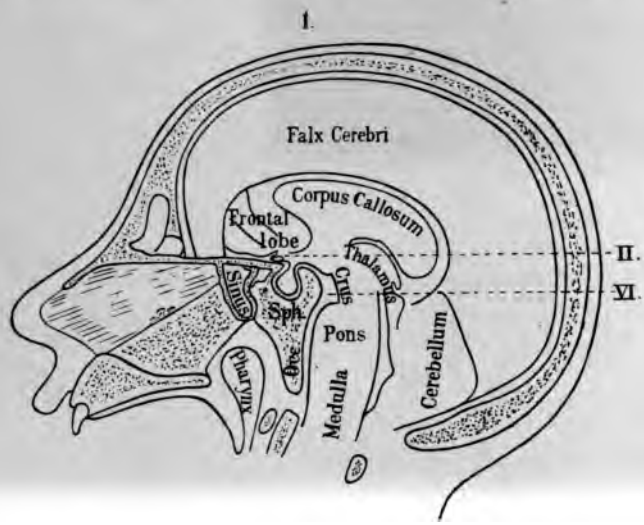

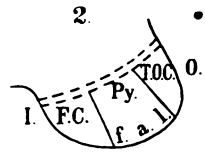

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DESCRIPTION OF PLATE XIII.

To illustrate Mr. Spencer's Case of Bullet Wound of the Brain.

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upwards as if to receive something into it. Thus the shoulder is retracted, the elbow extended, the forearm overpronated, the wrist flexed, the fingers and thumb extended.

Sensation is normal. There is no wasting of muscle, nor cutaneous nerve lesions. He has pain in the muscles of the arm, extending downwards, especially to the middle finger, whenever the clonic movements are in excess, to diminish which he holds his right arm forwards across his chest with his left.

Right leg: He walks with a stiff knee owing to the tonic spasm of the thigh muscles, but there is no loss of voluntary movement, for he can walk for two hours at a stretch, and go upstairs without assistance, but finds some difficulty in going downhill. Sometimes the tonic spasm is increased in extent, so that there is flexion at the hip and extension at the ankle, but not clonic movements.

The present position of the bullet is seen by examining the skiagram. An antero-posterior skiagram shows the bullet just to the left of the middle line, and the lateral skiagram (Plate XIII), when compared with the diagram (1) of the base of the skull, shows that the bullet is situated a little behind the posterior clinoid process. In its course the bullet has practically divided the left optic nerve (see Diagram 1, II), leaving only sufficient fibres to allow of sufficient light and of the pupil reflex. It has also injured some of the fibres of the chiasma, or left optic tract, which cross at the chiasma to go to the right eye, as shown by the limitation of the field of vision. The bullet has injured the left sixth nerve, doubtless about the spot where it enters the dura mater (Diagram 1, VI), behind and below the posterior clinoid process and internal to the fifth nerve, as shown by the loss of power in the left external rectus muscle. Also it has injured the cilio-spinal sympathetic fibres, doubtless close to the entrance of the internal carotid into the skull, as shown by the persistence of a contraction of the pupil in spite of the eye being blind, and by the loss of the sympathetic reflex. The third nerve has escaped injury; its fibres to the upper lid, to the eyeball, to the iris and ciliary muscle are intact. The course of the bullet was evidently internal to the carotid and cavernous sinus, for there is no sign of an arterio-venous aneurysm, although some orbital or other small vein was injured at the time, and caused the left orbital hæmorrhage. Likewise the bullet is clearly internal to the root and ganglion of the fifth nerve, so that no disturbance of sensation has been set up.

The most striking effect of the injury is an irritative lesion of the descending pyramidal tract, corresponding with the position of the bullet a little behind the left posterior clinoid process. Diagram 1 shows that the bullet must be here in relation with the left crus cerebri. Diagram 2 shows the division of the fibres in the crusta on the antero-inferior aspect of the crus. The centre of the crusta is occupied by the pyramidal (*Py.*) fibres, the middle fibres of which (*a.*) pass to the arm, the inner (*f.*) to the face, and the outer (*l.*) to the leg. The stress of the lesion has been on the middle, the arm fibres, those for the face and leg on either side being less involved.

XXXVII.—*Normal Thoracic Resonance in Left-handed Persons.* By Sir Hugh Beevor, Bart., M.D. *Exhibited April* 26, 1901.

THESE cases are brought before the Society, as they afford an explanation very positively that the lesser resonance on the right chest in right-handed persons is due to muscular development or to the greater use of the muscles of that side.

In the absence of emphasis upon this point in text-books mistakes are still often made and disease wrongly suspected. I find Mr. W. H. Brazil, in *Brit. Med. Journ.*, October 26, 1889, has emphatically called attention to this point in left-handed persons.

When lung percussion resonance is tested to its utmost limit on either side of neck, the resonance does not extend so high on the right side in a large proportion of people. It is also normal in these persons to find resonance diminished down to the first interspace.

It may be noted in these cases pulmonary resonance does not rise so high above the left clavicle as above the right; the diminished resonance or higher note to be noted below clavicle is distinct in the younger man.

I have found no reason to suspect either is the subject of disease; they repeat former observations I have made upon left-handed persons.

XXXVIII.—*Unusual Case of Obesity in a Child.* By G. F. Still, M.D. (introduced by Dr. Dalton). *Exhibited April* 26, 1901.

MARTIN R., æt. 7¼ years, was very thin one year ago. He was then taken to Ramsgate for six weeks, and whilst there rapidly became fatter; since that time there has been a steady increase of fat, and during the last two months he has been somewhat short of breath. Within the last three weeks the lower part of abdomen and the upper part of thighs have shown irregular bluish-red lines where the skin is thinned, and shiny with dilated venules, evidently analogous to "lineæ gravidarum."

One month ago he awoke one morning quite blind, but otherwise in his usual health; the blindness lasted forty-eight hours. Sight was normal when the child awoke on the third morning. He complains occasionally of some pain "like pins and needles" over the vertex of the head, but otherwise has had no pain anywhere. At the age of three years and a quarter the child had some severe illness, which began with convulsions, and was called "peritonitis." It lasted four months, and since then the abdomen has always been rather large. With the exception of measles at four years he has had no other illness.

One brother has pulmonary tuberculosis, and there is some more distant history of tuberculosis. The other members of the family are all said to be thin.

Present condition.—General appearance is striking, owing to great accumulation of subcutaneous fat; the cheeks are ruddy and bulging, and over the rest of the body there is a great excess of subcutaneous fat in all parts where it is normally present. In the supra-clavicular fossæ, and over the vertebra prominens behind, there are collections of fat, perhaps more marked than elsewhere, but otherwise the increase of fat is uniform and diffuse. The abdomen is large —its prominence is due chiefly to fat in its wall; and over the lower part of abdomen, as also on the upper part of thighs, bluish-red lines, about half an inch wide at some parts and three or four inches long, are seen as described above. The child's weight is 4 st. 11½ lbs., his height is 42½ inches.

There is some hair over the pubes, said to have been

present two years; the child's voice is not gruff. Right testicle normal; left not felt.

Thyroid gland cannot be felt; it is certainly not enlarged.

Lungs and abdominal viscera seem normal.

Heart shows nothing abnormal except an occasional slight irregularity of rhythm and marked accentuation of the second sound in aortic area.

XXXIX.—*Sequel of a case of Myxœdema which was under observation at intervals for twenty-three years.* By Sir Dyce Duckworth, M.D. *Exhibited April* 26, 1901.

S. M., æt. 56. This patient began to manifest symptoms of myxœdema in 1878, and came under my care as an out-patient at St. Bartholomew's Hospital in 1879. I recorded the particulars of her case in the Clinical Society's *Transactions* for that year.* I lost sight of her for some time, but she subsequently came into Elizabeth Ward under my care in 1893, weighing 14 st. 4 lbs., and remained for three months under observation. Her weight was reduced to 10 st. 5½ lbs. I have seen her at long intervals since, and she has maintained a good general condition of health for many years under the influence of thyroid medication. Not feeling well, she sought re-admission to the hospital, and came again under my care in John Ward on April 15, 1901. Her case was one of the earliest brought before the notice of this Society, and at that time the intimate nature of the malady was not ascertained. We had not then heard of *cachexia strumipriva*, and Horsley's experimental work had not been achieved. The case is so remarkable and satisfactory that it calls for a record of its progress for its own sake, and as an encouragement to persevere with the line of treatment now so fully established. I therefore subjoin a report on the present condition of this woman, and have endeavoured to record all the facts that appear to be of interest in connection with the disorder.

The influence of thyroid treatment was more marked in this instance than in any that I have witnessed. The patient

* Vol. xiii, p. 12, 1880.

was growing steadily worse, becoming languid, helpless, and hebete, and becoming a burden to herself and her family. Within a few months she regained her juvenile figure, and became bright and useful once more. Her third, rickety child died, but she again became pregnant, and has borne three more children.

Her present complaint is chiefly of occipital and vertical headache, and there is no obvious relapse of the myxœdematous state. There are no morbid indications in the thorax or abdomen. Some additional moles have appeared on various parts of the body. The blood-corpuscle count is good = red 4,660,000, white 7600. Hbg. 88 per cent. Colour index ·98. Temperature 97°.

The urine is void of albumen and sugar, depositing lithates, and containing urea 4·4 per cent., and later 3·2 per cent., 1·030, acid.

The knee-jerks are present but sluggish. Fine movements of fingers are good.

The electrical, muscular, and nervous reactions are normal. Cerebration not slow. She has taken thyroid elixir (f.ʒj = one thyroid gland [sheep's]) in f.ʒss doses *per diem* for long periods, then less and less to the amount of f.ʒij in a week for years. Her present weight is 10 st. 13 lbs. No supraclavicular fatty masses to be felt.

XL.—*Retro-collic Spasm.* By C. E. Beevor, M.D. *Exhibited April* 26, 1901.

THOMAS O., æt. 44, engine fitter, married, three children. Onset gradual.

Supposed cause: father died just before onset, and patient much upset by this.

Five years had occasional attacks of muscular rheumatism in lumbar region, otherwise no previous illness.

Present illness began two years ago with lumbago. Sharp pains, which became later of dull aching character.

In 1899 had spasm of muscles round hip, with hardness and stiffness there, which gradually passed up to his shoulders, and by September, 1900, he had marked retraction of head and neck. He has done no work since June, 1900.

Present state.—As patient lies resting at full length there is no spasm, but on sitting up, and especially on walking, he gets spasm of the muscles of neck, so that the cervical spine is extended concave backwards and the chin depressed, so that the vertical mental line falls behind the pomum Adami: there is no extension of the head on the spine. The extensions of the head, the complexus, and the trapezius do not contract, and the muscles affected are probably the deep extensions of the cervical spine; but the complexus can be voluntarily made to contract by extending the head against resistance. The sterno-mastoids are tightened up, but apparently are not spasmodically contracted. On walking the erector spinæ is spasmodically contracting, producing lordosis and throwing the chest forwards.

The spasm ceases if the head is allowed to fall forwards fully flexed, or if any support is given to the head from behind, or on lying down.

Treatment consisted in Weir-Mitchell treatment.

Suspension from the head three times a day for ninety seconds; strychnine, cannabis indica, bromides, belladonna, and the constant current.

XLI.—*A case of Lymphadenoma with Growths in the Skin.* By Lee Dickinson, M.D. *Exhibited April* 26, 1901.

THE case is that of a boy, P. P., æt. 15, the son of a labourer, who has suffered from enlargement of the lymphatic glands since July, 1899. The first glands noticed to be enlarged were the cervical, at which time the general health was good. In November, 1899, he came into St. George's Hospital under the care of Dr. Isambard Owen, when, besides considerable enlargement of the cervical and submaxillary glands, the inguinal also were affected. Some carious teeth were extracted without, however, much effect upon the neighbouring glands, and he was put upon treatment by arsenic internally in the form of Donovan's solution, ♏v ter die. The arsenic was taken almost without intermission, and mainly in the same form, for more than six months, a great part of which time he spent in our Con-

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DESCRIPTION OF PLATE XIV.

To illustrate Dr. Lee Dickinson's Case of Lymphadenoma.

valescent Hospital at Wimbledon, where he came under my care. In spite of the arsenic the disease made steady progress. Thus in February, 1900, the enlargement had extended to the axillary and clavicular glands of each side, and probably also to the mediastinal, as suggested by impaired resonance on the sternum, dyspnœa on exertion, and slight dysphagia. Somewhat later appeared symptoms of pelvic and abdominal pressure—cramps in the thighs, and vague discomfort about the stomach; and by the beginning of June the boy's condition was decidedly threatening, although he was not confined to bed. About this time there appeared over the right side of the chest, from above the clavicle to below the nipple and over the upper part of the scapula, a copious deposit of little growths in the skin. The nature of this skin affection being at first doubtful the arsenic was stopped, but all suspicion as to its arsenical origin was removed in the course of a month by increase in the number and size of the deposits, and by the nipple becoming involved and fixed.

The boy was then sent to the Royal Sea Bathing Hospital at Margate, where he remained for three months, and derived considerable improvement in general health. He spent the winter in Hertfordshire, not under medical care; and finding himself growing thinner and weaker he applied again at St. George's, and was re-admitted under my care at the beginning of the present month (April, 1901).

He has grown in height since last July, and is much improved in appearance owing to diminution of the cervical swelling, but he is now very thin and notably anæmic. There is no evidence of pressure within the thorax or abdomen, and the axillary glands are smaller than they were; but the glandular affection is more general. Substantial tumours can be felt above the clavicles and in the occipital region, and the trochlear glands are slightly enlarged. The inguinal glands are larger than before, and above the groins and over the lower abdomen are numerous little nodules in the skin which can be felt rather than seen. A few such nodules are found even above the umbilicus and along the sides of the thorax, and others can be felt in the arms for a few inches above the internal condyles. These nodules probably represent an early stage of the conspicuous skin growths which are seen in the right clavicular and pectoral region, and which are all that remain of the far more diffuse, though individually less conspicuous deposits of nine months ago. These growths are mainly of a reddish colour and no little prominence, but

in more than one place the tint is yellow, and the surface depressed—appearances which suggest that involution in the shape of fatty degeneration is taking place. A small piece has been snipped out of the largest skin growth, and kindly examined under the microscope by Dr. Rolleston, who confirms the clinical view that the growth is lymphadenoma.

The abdomen is full, but neither the liver nor spleen is definitely enlarged. The blood shows simple anæmia with moderate leucocytosis; the leucocytes number 12,000 per cm., the several classes preserving their proper proportions.

The urine is normal, and there are no signs of tubercle in the lungs or elsewhere.

Certain bony changes of doubtful significance perhaps deserve mention. The upper ends of both tibiæ are larger than normal, and the inner tuberosities are tender on percussion. On the anterior surface of the left tibia is a large flat node, not in the least tender. If these bony changes are to be taken as evidence of syphilis, they are certainly unsupported by manifestations elsewhere. The teeth, hair, and eyes show nothing characteristic. Scars are absent, as also is pigmentation, except a brown patch on the front of the left knee and a few freckles on the thigh, attributed to scorching from sitting over the fire.

The temperature is not quite regular, occasionally rising above 100°, and the pulse is generally above 100°; but the boy is in good spirits and no discomfort, and his general condition may be stated as fairly satisfactory considering the probably hopeless nature of the disease.

XLII.—*Congenital Morbus Cordis, ? patent Ductus Arteriosus.* By Percy Kidd, M.D. *Exhibited April* 26, 1901.

SUSAN G., æt. 21, cigar maker. Cyanosis slight, especially of conjunctiva; no clubbing.

Heart enlarged, right side mainly. Faint diastolic thrill in second left space. Second sound everywhere very loud; maximum intensity at third left cartilage. At this point a short diastolic murmur following second sound audible at times. First sound everywhere feeble and generally doubled.

MAOU

DESCRIPTION OF PLATE XV.

To illustrate Dr. Kidd's Case of Myxœdema.

History.—Attacks of cardiac pain from childhood. Dyspnœa for three years. No rheumatism, scarlet fever, or chorea.

XLIII.—*Myxœdema in a girl æt.* 15. By Percy Kidd, M.D. *Exhibited April* 26, 1901.

On admission, February 27th, 1901, the patient presented the typical appearances of myxœdema—sallow anæmic colour, bright flush over the malar bones, almond-shaped eyes, and puffy, swollen condition of the skin of the face.

Skin harsh and dry, with exception of palms, which were moist.

Thyroid gland could just be felt.

Firm fatty masses in both supra-clavicular fossæ.

Expression stolid, but intelligence good. When she left school, at the age of thirteen, had reached fourth standard.

The history suggested a congenital origin, though the appearances were not those of cretinism.

The child did not walk till the age of two and a half years, and used to pass urine and stools in bed up to the age of six years.

At two years began to get very fat.

Had always been sluggish, and not nearly as bright as the other children.

For the last twelve months increasing hebetude and frequent headaches.

Treatment with thyroid extract produced rapid improvement, and the appearance of the child is no longer characteristic of myxœdema.

XLIV.—*A case of Chronic Hypertrophic Osteo-arthropathy.* By A. B. Soltau, M.B. (introduced by Percy Kidd, M.D.). *Exhibited April* 26, 1901.

Annie K., æt. 36, married, was admitted into the London Hospital under the care of Dr. Francis Warner, to whom I am indebted for permission to publish these notes.

She was last quite well eighteen months ago, when she

had an attack of influenza, which confined her to bed for several days. She then noticed her knees were swelling, and they became very painful. Then her hands became large, and also were painful. Twelve months ago the feet and ankles became affected.

She has wasted and lost 4 st. 4 lbs. in weight. No history of any previous illnesses was obtainable, nor has she suffered from cough. The family history has no points of interest.

On admission: patient is obviously wasted and has a drawn expression. The thoracic and abdominal viscera are healthy. There is no trace of pulmonary complaint. The hands are large, and the fingers marked[illegible]ibbed. The superficial veins and radial arteries are large. The nails are curved over the ends of the phalanges. There is effusion into the wrist-joints. There is considerable thickening round the mid-phalangeal joints. The metacarpus, phalanges, and lower end of radius and ulna show bony thickening. As evidence of the increase in size of the hands, patient used to take No. 7 in gloves and now cannot obtain any large enough. There is no grating in any of the joints, which are rather tender on examination. The toes are affected in the same way as the fingers, though to a less degree. There is considerable effusion into both ankles and both knee-joints. Skiagraphs show thickening of the tibia and fibula, and the lower end of the shaft of the femur, also of the phalanges of the feet. The effusion into the knee-joints is sufficient to permit of lateral movement. The spine is kyphotic. There is marked wasting of the erector spinæ, quadriceps extensors, and small muscles of the hands. The muscles do not give the reaction of degeneration. The face is thin. There is no enlargement of the facial bones, nor any exostoses on the skull, which measures twenty-two inches in circumference. The fundi oculorum are normal, and there is no diminution in the field of vision. The urine is normal in quantity and constituents. The superficial and deep reflexes are normal.

The case resembles those described by Marie as pulmonary osteo-arthropathy. This case is of interest in that no pulmonary signs are present, as in some few cases already described. It is, I believe, the first case described in which the patient dates the commencement of the condition to an attack of influenza.

The radiographs show very well the bone changes, the thickening being along the shafts of the bones and not confined to the ends, and also the absence of exostoses.

DESCRIPTION OF PLATE XVI.

To illustrate Mr. Soltau's Case of Osteo-arthropathy.

FIG. 1.

FIG. 2.

Bale and Danielsson, Ltd.

DESCRIPTION OF PLATE XVII.

To illustrate Mr. Soltau's Case of Osteo-arthropathy.

DESCRIPTION OF PLATE XVIII.

To illustrate Mr. Soltau's Case of Osteo-arthropathy.

FIG. 1.

FIG. 2

Bale and Danielsson, Ltd.

XLV.—*Inveterate Ulceration of both Hands.* By J. HUTCHINSON, jun. *Exhibited April* 26, 1901.

THE patient was a letter sorter æt. 36; there was nothing in the personal or family history to point to tubercular tendency.

In April, 1900, a red spot appeared on the back of the right hand, which ulcerated and continued in the form of a sore with firm slightly raised edges for twelve months. The ulcer at one time was three to four inches in diameter, covering the ulnar portion of the dorsum. There was a thin discharge from it, but nothing suggesting gummatous material. The edges were nowhere undermined.

In March, 1901, a more superficial ulcer developed on the back of the left hand, just below the ulnar head.

The patient was most thoroughly treated in private, at the Royal Free Hospital, and at the London Hospital, but although partial cicatrisation occurred the skin always broke down again. From its method of commencing and appearance it was suspected to be lupus, and was once scraped without benefit. There was a history of the patient having had a chancre, but none of secondary syphilis. Iodides were tried for long, and mercurial ointments applied. The edge of the ulcer at parts suggested rodent ulcer, and with this view the Röntgen rays were given a thorough trial by Dr. J. H. Sequeira, with temporary improvement.

Mr. Hutchinson regarded the ulceration as being primarily due to poor circulation and nutrition of the skin in a part which was especially exposed to cold and changes of temperature. In favour of this was the history of cold hands and feet in the patient, and the ulcer of the left hand in an almost symmetrical position tended to corroborate it. There is no part of the body in which the skin is more poorly supplied with arterial blood and more liable to starvation than the back of the hands.

The result of treatment founded on this view was conclusive. The ulcer, which had entirely resisted the remedies for a year, healed soundly in three weeks under the following measures. The patient was kept in hospital, fed well, the arm and hand enveloped in cotton wool and kept at rest on a

splint. A stimulating ointment (red oxide of mercury) was applied, but no medicines were given.

Both hands were well in three weeks, and the patient was recommended to wear warm gloves as a rule.

These cases are interesting from the suspicions as to malignant disease, syphilis, or tubercle that they give rise to, and from the extreme obstinacy of the ulceration. The patient had been more or less laid up and prevented from working for a year. The term "asthenic ulcer" is very applicable to the condition.

XLVI.—*Dislocation of the Patella.* By Charters Symonds, M.S. *Exhibited April* 26, 1901.

THE left patella is dislocated outwards when the knee is flexed. On extension it lies rather to the outer side of the usual position. The knee is strong, he can kneel, and walk quickly. When younger he used to stumble easily.

The limb being useful it was not proposed to undertake any treatment.

XLVII.—*Hæmorrhagic Gummatous Myositis.* By Charters Symonds, M.S. *Exhibited April* 26, 1901.

HENRY L., aged about 45. Exhibits large irregularly formed masses in the extensor muscles of the right thigh. There is a history of injury, followed in a few days by swelling. When first seen the swelling was very large, the skin involved and red in patches, with effusion into the knee. Under iodide of potassium rapid improvement ensued up to a certain point. It is suggested that there was originally specific myositis, the rapid increase being due to hæmorrhage.

XLVIII.—*Knee-joint Eight Years after Erasion.* By CHARTERS SYMONDS, M.S. *Exhibited April* 26, 1901.

FLORENCE W., æt. 19. The case is exhibited to show the condition of a knee-joint eight years after erasion. The movement is free to a right angle, but there is a good deal of weakness. The question raised by the case is whether the patient is as well off as after excision. The exhibitor suggests that the stiff leg would probably be preferable to the present condition.

The patient is able to discharge all the duties of a general servant, including the carrying of trays upstairs. She can step from a chair to the floor, landing on the affected side. While the usefulness is exceptional there is at times some aching and heat in the joint. As to the question of the disease being of a syphilitic character and not tuberculous, it may be added, that the condition of the joint when excised showed the ordinary appearances of tubercle. Treatment by rest and pressure, with mercury internally, in addition to other usual remedies, had been carried out for several years.

XLIX.—*Sequel* to a case of Myasthenia gravis pseudo-paralytica (Strümpell's Asthenic Bulbar Paralysis).* By LEONARD GUTHRIE, M.D.

THIS patient, a girl æt. 23, was exhibited before the Society on February 23, 1901. Her symptoms (of two years' duration) were difficulty in articulation, mastication, and deglutition occurring mostly after fatigue, with general weakness following slight exertion. She showed paresis of the orbicularis oculi and oris, and of the transverse lingual muscle. A typical "myasthenic" reaction to faradism was demonstrated in the muscles.

* The case has been published in full in the *Lancet* for February 9, 1901.

Progress.—At the end of three months' observation the general condition had greatly improved. There was now no actual inability to swallow nor regurgitation. Speech only became indistinct after reading aloud for several minutes. The right eye could be almost closed, and resistance to passive elevation of the eyelids was more apparent than it had been. The mouth looked smaller and less flabby. She felt stronger, and had gained three and a half pounds in weight. Treatment had been by rest and the administration of liquor strychniæ.

Subsequent history.—She left the hospital at the beginning of May, 1900, and went to Tunbridge Wells, where she remained until a severe relapse occurred on June 15. Her mother believed that the relapse was due to mental trouble. An engagement to marry had been broken off, and her *fiancé* had gone to the war in South Africa. When seen on June 19 her condition was much worse than it had ever been. She could not swallow water even in sips. Her speech was quite unintelligible, and the palatal movements were more sluggish than before. Her mouth was full of glairy mucus and saliva, which she could neither swallow nor eject. Her neck muscles were weakened, so that her head fell backward if she inclined it beyond the centre of gravity, unless she supported it with her hands. She was very depressed and tearful. On July 3 the weakness of the neck muscles had passed off. On the previous day she had choked whilst trying to swallow a small piece of bread and butter. She could neither get it up nor down, but her mother succeeded in dislodging it with her finger. On the whole she seemed rather better. She was not seen alive at hospital again, but Dr. Edmund Holland, of Regent's Park, has kindly given particulars of the conclusion of the case.

On July 10 she started for a short walk, but soon returned, complained of great fatigue, and finally took to her bed. On the night of the 11th Dr. Holland was called to see her, and found her suffering from a bad attack of dyspnœa. She was fighting hard for breath, and he thought that she was dying. The attack, however, passed off in about an hour, after which she slept quietly. The next morning she was breathing easily, but she could not sit up in bed without assistance. She had only taken about a pint of liquid nourishment in twenty-four hours for several days, and deglutition was getting more and more difficult, so he ordered her to be fed by nutrient enemata only. On July 14 general

weakness was much more marked. After talking a little her voice became reduced to a whisper. On the 15th the diaphragm was apparently paralysed, as she could not cough to clear her throat of mucus without pressing her hands on the lower ribs and epigastrium. She died quietly about 3 A.M. on the 16th. Consciousness was maintained until an hour before death, which was probably due to respiratory failure and gradual exhaustion.

Necropsy.—A *post-mortem* examination was made thirty hours after death. The body was slight in build, but not emaciated. Rigor mortis was well marked. All the internal organs seemed healthy, and also the brain and spinal cord.

Microscopical examination.—Dr. H. Campbell Thomson reports as follows: "The cord, medulla, and portions of cortex were hardened in formalin (5 per cent.), cut with a freezing microtome, and stained with carbol-thionin. The cells of the hypoglossal and glosso-pharyngeal nuclei for the most part appeared normal; in some, the chromatophile granules were not differentiated, and formed a homogeneous staining, which, however, was not darker than normal. There was no loss of chromatophile bodies noticed in any of the cells. Cells from the spinal cord at the level of the third cervical roots showed similar changes. No other changes were detected either in the cells or in the other tissues of the cord. Examination of the cortex and of the nerve-trunks gave negative results." Dr. Thomson considers the changes described as merely due to slight variations in staining, and that the results of examination as a whole are negative. Thus the most modern methods of investigation only confirm the accuracy of Sir Samuel Wilks' description of the first case on record in this country, as one of "bulbar paralysis without anatomical changes."

The motor-nerve endings in muscles were not examined in this case because portions of muscles removed for that purpose were unfortunately lost. Yet I venture to think that examination of them would probably have been also negative, for the variation in symptoms shows that the motor end plates or terminals in muscles, the most delicate portions of the neurons, cannot be permanently disorganised. This is implied by the descriptive term "pseudo-paralytica," which forms the sub-title of the disease. Myasthenia gravis is, in fact, a condition of toxæmia. The toxin, whatever it be, appears to have a selective and narcotic action on the motor neurons as a whole, and when the neurons attacked are vital,

death is the result, in accordance with the virulence and intensity of the poison at work. Of the nature and origin of the toxin causing myasthenia, little or nothing is known. It is probably endogenous rather than exogenous, because the disease does not occur in epidemics or attack members of the same family simultaneously. It cannot be traced to any definite antecedent. The supposed exciting causes have been so many and so various, yet so often altogether absent, that we cannot attribute to them any important part in the causation of the disease beyond that of weakening the patient and rendering him susceptible.

INDEX.

PAGE

Abdominal section for ascites: result (N. Dalton, M.D.) . . 5
——— ——— in treatment of ascites (L. A. Bidwell) . . . 171
Abscess, sub-diaphragmatic, caused by perforating gastric ulcer (N. Dalton, M.D.) 165
Actinomycosis, acute (Sir D. Duckworth, M.D., and H. Marsh) . 1
——— in parotid region (R. Johnson) 235
Africa, South: enteric fever among the troops in South Africa (Orange River Colony) (H. H. Tooth, C.M.G., M.D.) . 81—99
Albuminuria wanting in case of nephritis (W. P. Herringham, M.D.) 34
Anæmia, pernicious: subacute combined degeneration of spinal cord in association with (F. E. Batten, M.D.) 56
Aniline oil, poisoning from external use (StC. Thomson, M.D.) . 156
Ankylostomiasis, case of (W. J. Tyson, M.D.) 37
Antenatal treatment of hæmophilia (W. H. B. Brook, M.D.) . 153
Aorta: regurgitation in, probably due to syphilis (C. W. Chapman, M.D.) 64
——— ——— apparently due to rupture of healthy valve (L. E. Shaw, M.D.) 220
Arm, see *Forearm.*
Army in South Africa: see *Troops.*
Arthropathy: see *Osteo-arthropathy.*
Ascites: cases treated by abdominal section (L. A. Bidwell) . 171
——— results of abdominal section in (N. Dalton, M.D.) . . 5
——— simulated by pancreatic cyst of enormous size (S. Phillips, M.D.) 53
Auricle: extreme dilatation of left auricle of heart (Isambard Owen, M.D., and W. J. Fenton, M.D.) 183

Bath: electric light and heat bath (F. C. Eve, M.D.) . . . 225

PAGE

BATTEN (Frederick E., M.D.), a case of subacute combined degeneration of the spinal cord in association with pernicious anæmia 56
——— a case of localised muscular rigidity 219
BEEVOR (C. E., M.D.), a case of hæmorrhage into the spinal cord (shown by W. A. Marris, M.D.) 212
——— a case of multiple fibro-neuromata (with examination of an excised tumour by Dr. James Collier) . . . 226, 227
——— retro-collic spasm 245
BEEVOR (Sir Hugh, Bart., M.D.), normal thoracic resonance in left-handed persons 242
BIDWELL (Leonard A.), some cases of ascites treated by abdominal section 171
Bladder, calculus of, simulated by ureteral calculus (F. J. Steward) 149
BLAND-SUTTON (John), a case in which splenectomy was performed for a wandering spleen 31
Blood and vascular changes in case of cancer of stomach (W. Pasteur, M.D., and C. Price-Jones, M.B.) 160
BOYD (Stanley), oöphorectomy for recurrent mammary cancer . 214
Brain: bullet wound, showing injuries to optic nerve and pyramidal tract corresponding to course and present position of bullet (W. G. Spencer) 238
Breast, cancer of, treated by oöphorectomy (S. Boyd) . . . 214
——— carcinoma mammæ followed by multiple cutaneous carcinomatosis (H. D. Rolleston, M.D.) 206
BRIDGER (A. E., M.D.), a case of chronic lymphangitis (introduced by C. S. de Segundo, M.D.) 229
BROADBENT (Sir William H., Bart., M.D., F.R.S.), discussion on the epidemic of enteric fever among the troops in South Africa 115
Bronchocele, acute (S. Phillips, M.D.) 236
BROOK (W. H. B., M.D.), a case illustrating an attempt at the antenatal treatment of hæmophilia 153
BRYANT (J. H., M.D.), a paper on instantaneous radiography. By Edward W. H. Shenton (introduced by J. H. Bryant, M.D.) 76
——— a case of volvulus of the ileum with severe intestinal hæmorrhage 144
Bullet wound of brain showing injuries to optic nerve and pyramidal tract corresponding to course and present position of bullet (W. G. Spencer) 238
——— ——— transfacial (H. Batty Shaw, M.D.) . . . 210

PAGE

BURTON-FANNING (F. W., M.D.), a case of progressive myositis ossificans (shown by H. D. Rolleston, M.D., for F. W. Burton-Fanning, M.D.) 232

Calculus, ureteral, simulating vesical calculus (F. J. Steward) . 149
CALVERT (James, M.D.), a case of hæmato-porphyrinuria, not due to sulphonal, with a report on the urine by A. E. Garrod, M.D. 41
Cancer of breast treated by oöphorectomy (S. Boyd) . . . 214
——— of stomach, with unusual vascular and blood changes (W. Pasteur, M.D., and C. Price-Jones, M.B.) 160
Carcinoma of breast followed by multiple cutaneous carcinomatosis (H. D. Rolleston, M.D.) 206
Carcinomatosis, multiple cutaneous, after carcinoma mammæ (H. D. Rolleston, M.D.) 206
Cardiac sound: see *Heart* . . . . . .
CARR (J. Walter, M.D.), a case of myositis ossificans progressiva 199
CHAPMAN (Charles W., M.D.), a case of aortic regurgitation probably due to syphilis 64
Child, unusual case of obesity in (G. F. Still, M.D., introduced by Dr. Dalton) 243
CHURCH (Sir William, Bart., M.D.), discussion on the epidemic of enteric fever among the troops in South Africa . . 119
CLARKE (J. Jackson), a case showing the result of a plastic operation for the relief of rectal incontinence left after a modified Kraske's excision of the rectum for cancer . . 195
CLARKE (W. Bruce), a case of intractable ulcer of the forearm of undetermined origin 214
COLLIER (James, M.D.), examination of an excised tumour by Dr. James Collier, in Dr. C. E. Beevor's case of multiple fibro-neuromata 227
Convulsions occasioned by traumatic subdural hæmorrhage (R. Johnson and J. Risien Russell, M.D.) 179
Council: List of Officers and Council, 1901—1902 . . . xix
——— Report of the Council li
CROMBIE (Alexander, (M.D.), discussion on the epidemic of enteric fever among the troops in South Africa . . . 127
Curvature, extraordinary, of lower end of femur following epiphysitis (J. S. Richards, M.D.) 231
Cyst: cystic kidneys, case apparently congenital (N. Dalton, M.D.) 208
——— pancreatic, of enormous size, simulating ascites (S. Phillips, M.D.) 53

PAGE

DALTON (Norman, M.D.), the sequel to a case of hydatid of the liver: showing also the results of abdominal section in ascites 5
——— a case of perforating gastric ulcer, causing a subdiaphragmatic abscess, which burst through the diaphragm into the lung 165
——— a case of cystic kidneys, apparently congenital. . . 208
Degeneration, subacute combined, of spinal cord, in association with pernicious anæmia (F. E. Batten, M.D.) 56
Dextrocardia, case probably congenital (A. M. Gossage, M.D.) . 220
Diaphragm: perforating gastric ulcer causing subdiaphragmatic abscess which burst through diaphragm into lung (N. Dalton, M.D.) 165
DICKINSON (W. Lee, M.D.), a case of lymphadenoma with growths in the skin 246
Dilatation, extreme, of left auricle of heart (Isambard Owen, M.D., and W. J. Fenton, M.D.) 183
——— of the heart, unusually great (J. P. Parkinson, M.D.) . 221
Dislocation of patella (C. J. Symonds) 252
DUCKWORTH (Sir Dyce), discussion on the epidemic of enteric fever among the troops of South Africa 109
——— sequel of a case of myxœdema which was under observation at intervals for twenty-three years 244
——— and MARSH (Howard), a case of acute actinomycosis . 1
Ductus arteriosus: ? patent ductus arteriosus regurgitation in case of congenital morbus cordis (P. Kidd, M.D.) . . . 238
——— ? patent ductus arteriosus in case of congenital morbus cordis (P. Kidd, M.D.) 248

Elbow, excision in peculiar and unexpected manner; case shown after three years (Rushton Parker) 216
Electric light and heat baths (F. C. Eve, M.D.) 225
Empyema of frontal sinus, chronic; radical cure (H. Tilley, M.D.) 201
——— right, complicating case of pneumococcal suppurating pericarditis (J. Fawcett, M.D., and F. J. Steward) . . 46
Enlargement of right foot (H. B. Robinson) 234
Enteric fever among the troops in South Africa, in the Orange River Colony (H. H. Tooth, C.M.G., M.D.) . . . 81—99
——— see also *Typhoid* fever.
Epiphysitis followed by extraordinary curvature of lower end of femur (J. S. Richards, M.D.) 231

PAGE

Erasion of knee-joint, case shown eight years after erasion (C. J. Symonds) 253
EVE (F. C., M.D.), a simple form of electric light and heat bath 225

Face: transfacial bullet wound (H. Batty Shaw, M.D.) . . 210
FAWCETT (John, M.D.) and STEWARD (F. J.), a case of pneumococcal suppurating pericarditis treated by incision and drainage; right lobar pneumonia; right empyema; death . 46
Femur: curvature, extraordinary, of lower end following epiphysitis (J. S. Richards, M.D.) 231
FENTON (W. J., M.D.) and OWEN (Isambard, M.D.), a case of extreme dilatation of the left auricle of the heart . . . 183
Fever (enteric), see *Enteric* fever.
——— intermittent essential fever of syphilis (late syphilitic pyrexia) (Campbell Williams) 28
——— (malarial): see *Malaria.*
——— (typhoid): see *Typhoid* fever.
Fibro-neuromata, multiple (C. E. Beevor, M.D.) 226
Foot, right, enlargement (H. B. Robinson) 234
Forearm: intractable ulcer of undetermined origin (W. Bruce Clarke) 214

GARROD (Archibald E., M.D.), a case of hæmatoporphyrinuria not due to sulphonal, by James Calvert, M.D. With a report on the urine by A. E. Garrod, M.D. 41
——— a case of peculiar affection of the joints. (Shown by Hugh Thursfield, M.D., for A. E. Garrod, M.D.) . . . 233
Gastric ulcer, perforated: thrombosis of inferior vena cava and of iliac and femoral veins, of acute onset and fatal termination, with symptoms resembling those of perforated gastric ulcer (S. Phillips, M.D.) 24
——— ——— perforating, causing subdiaphragmatic abscess (N. Dalton, M.D.) 165
GODLEE (Rickman J.), acute suppuration of the thyroid gland complicating typhoid fever 189
——— a case of injury to left optic nerve 237
Gonorrhœa: See *Rheumatism* (Gonorrhœal).
GOSSAGE (A. M., M.D.), a case of dextrocardia, probably congenital 220
Graves's disease: case of tachycardia (? Graves's disease) (S. Phillips, M.D.) 237
Growth of the left tonsillar region (StC. Thomson, M.D.) . . 203

PAGE

Growths in skin in case of lymphadenoma (W. Lee Dickinson, M.D.) 246
GUTHRIE (Leonard, M.D.), sequel to a case of myasthenia gravis pseudo-paralytica (Strümpell's asthenic bulbar paralysis) . 253

Hæmatoporphyrinuria not due to sulphonal (J. Calvert, M.D.) . 41
Hæmophilia, attempt at antenatal treatment (W. H. B. Brook, M.D.) 153
——— with disease of the knee-joint (P. Kidd, M.D.) . . . 205
Hæmorrhage into the spinal cord (case shown by W. A. Marris, M.D., for C. E. Beevor, M.D.) 212
——— severe intestinal, in case of volvulus of ileum (J. H. Bryant, M.D.) 144
——— subdural, traumatic, occasioning convulsions; successful operation (R. Johnson and J. Risien Russell, M.D.) . . 179
Hand: ulceration, inveterate, of both hands (J. Hutchinson, jun.) 251
Heart: diastolic cardiac sound causing spurious reduplication of second sound at apex (T. Stacey Wilson, M.D.) . . . 10
——— dilatation, unusually great (J. P. Parkinson, M.D.) . . 221
——— ——— extreme, of left auricle (Isambard Owen, M.D., and W. J. Fenton, M.D.) 183
——— disease, congenital; case of congenital morbus cordis (?) patent ductus arteriosus regurgitation (P. Kidd, M.D.) . 238
——— ——— congenital morbus cordis: (?) patent ductus arteriosus (P. Kidd, M.D.) 248
——— malposition: See *Dextrocardia.*
Heat and electric light bath (F. C. Eve, M.D.) 225
HERRINGHAM (W. P., M.D.), a case of nephritis without albuminuria; death in six weeks; no scarlatina; autopsy . . 34
HILLIER (Alfred P., M.D.), discussion on the epidemic of enteric fever among the troops in South Africa 133
HORTON-SMITH (Percival, M.D.) discussion on the epidemic of enteric fever among the troops in South Africa . . . 135
HUTCHINSON (Jonathan, jun.), inveterate ulceration of both hands 251
Hydatid of liver; sequel to case (N. Dalton, M.D.) . . . 5
Hygroma: cystic hygroma of neck, probably congenital (StC. Thomson, M.D.) 231

Ileum: volvulus of ileum with severe intestinal hæmorrhage (J. H. Bryant, M.D.) 144

PAGE

Intestine, hæmorrhage of, severe, in case of volvulus of ileum (J. H. Bryant, M.D.) 144

JAMESON (Surgeon-General James, C.B.), discussion on the epidemic of enteric fever among the troops in South Africa . 100
Jaw, lower: central sarcoma; case shown three and a half years after enucleation (C. J. Symonds) 213
JOHNSON (Raymond), a case of actinomycosis in parotid region . 235
——— and RUSSELL (J. Risien, M.D.), traumatic subdural hæmorrhage, occasioning convulsions on the sixth day after the injury, and successfully treated by operation . . . 179
Joints: a case of peculiar affection (shown by Hugh Thursfield, M.D., for A. E. Garrod, M.D., with report of Sub-Committee) 233

KIDD (Percy, M.D.), a case of hæmophilia, with disease of the knee-joint (with radiographs) 205
——— a case of congenital morbus cordis. (?) Patent ductus arteriosus regurgitation 238
——— congenital morbus cordis ? Patent ductus arteriosus . 248
——— myxœdema in a girl æt. 15 249
——— a case of chronic hypertrophic osteo-arthropathy (by A. B. Soltau, M.B., introduced by Percy Kidd, M.D.) . . 249
Kidneys, cystic, case apparently congenital (N. Dalton, M.D.) . 208
Knee-joint: disease of knee-joint in case of hæmophilia (P. Kidd, M.D.) 205
——— eight years after erasion (C. J. Symonds) 253

Left-handed persons, normal thoracic resonance in (Sir H. Beevor, Bart., M.D.) 242
Leg: symmetrical tumours of legs (J. S. Richards, M.D.) . . 227
Leucocythæmia, spleno-myelogenic, subsequent to attack of malarial fever (D. Nabarro, M.D.) 70
Lips, ulceration: intractable ulceration of lips and palate (G. R. Turner) 204
Liver, hydatid: sequel to case (N. Dalton, M.D.) 5
LONGHURST (Arthur E., M.D.), discussion on the epidemic of enteric fever among the troops in South Africa . . . 137
Lung: subdiaphragmatic abscess bursting through diaphragm into lung (N. Dalton, M.D.) 165
Lymphadenoma: case with growths in the skin (W. Lee Dickinson, M.D.) 246
Lymphangitis, chronic (A. E. Bridger, M.D.) 229

PAGE

MACPHERSON (Major), discussion on the epidemic of enteric fever among the troops in South Africa 110
Malaria: malarial fever followed by pronounced spleno-myelogenic leucocythæmia (D. Nabarro, M.D.) 70
MARRIS (W. A., M.D.) (for C. E. BEEVOR, M.D.) a case of hæmorrhage into the spinal cord 212
MARSH (Howard) and DUCKWORTH (Sir Dyce, M.D.), a case of acute actinomycosis 1
Members of the Society xxiii
——— honorary members xxi
——— foreign honorary members xxii
Muscles, localised rigidity (F. E. Batten, M.D.) 219
Myasthenia gravis (J. S. Risien Russell, M.D.) 216
——— ——— pseudo-paralytica (Strümpell's asthenic bulbar paralysis) sequel to case (L. Guthrie, M.D.) 253
Myositis, hæmorrhagic gummatous (C. J. Symonds) . . . 252
——— ossificans progressiva (J. W. Carr, M.D.) 199
——— ——— ——— (case shown by H. D. Rolleston, M.D., for F. W. Burton-Fanning, M.D.) 232
Myxœdema in a girl æt. 15 (Percy Kidd, M.D.) 249
——— sequel to case under observation for twenty-three years (Sir D. Duckworth, M.D.) 244

NABARRO (David, M.D.), a case of pronounced spleno-myelogenic leucocythemia subsequent to an attack of malarial fever (introduced by George Thin, M.D.) 70
Neck: cystic hygroma of, probably congenital (StC. Thomson, M.D.) 231
——— retro-collic spasm (C. E. Beevor, M.D.) 245
Nephritis without albuminuria; no scarlatina; death in six weeks (W. P. Herringham, M.D.) 34
Nerve, optic: case of injury to left optic nerve (R. J. Godlee) . 237
——— ——— injury to, in case of bullet wound of brain (W. G. Spencer) 238
Neuromata: see *Fibro-neuromata.*
Nodules, subcutaneous, in case of rheumatic periostitis (H. Thursfield, M.D.) 68

Obesity in a child, unusual case (G. F. Still, M.D., introduced by Dr. Dalton) 243
Officers of the Society xix

PAGE
Oöphorectomy for recurrent mammary cancer (S. Boyd) . . 214
Orange River Colony: enteric fever among the troops in South Africa, Orange River Colony (H. H. Tooth, C.M.G., M.D.) . 81-99
Osteo-arthropathy, chronic hypertrophic (A. B. Soltau, M.B., introduced by Percy Kidd, M.D.) 249
OWEN (Isambard, M.D.) and FENTON (W. J., M.D.), a case of extreme dilatation of the left auricle of the heart . . . 183

Palate, ulceration: intractable ulceration of lips and palate (G. R. Turner) 204
Pancreas, cyst of, simulating ascites (S. Phillips, M.D.) . . 53
Paralysis, Strümpell's asthenic bulbar (myasthenia gravis pseudo-paralytica), sequel to case (L. Guthrie, M.D.) . . . 253
PARKER (Rushton), a case of elbow three years excised in a peculiar and unexpected manner for stiffness following fracture; unusually strong result 218
PARKINSON (J. Porter, M.D.), a case of unusually great dilatation of the heart 221
Parotid region, actinomycosis in (R. Johnson) 235
PASTEUR (W., M.D.) and PRICE-JONES (C., M.B.), a case of cancer of the stomach with unusual vascular and blood changes . 160
Patella, dislocation of (C. J. Symonds) 252
PEARSON (S. Vere, M.D.) and ROLLESTON (H. D., M.D.), generalised scleroderma with Raynaud's disease 215
Pericarditis, pneumococcal, suppurating, treated by incision and drainage: death (J. Fawcett, M.D., and F. J. Steward) . 46
Periostitis, rheumatic, with subcutaneous nodules (H. Thursfield, M.D.) 68
PHILLIPS (Sidney, M.D.), a case of thrombosis of the inferior vena cava and of the iliac and femoral veins, of acute onset and fatal termination, with symptoms resembling those of perforated gastric ulcer 24
——— a case of fatal pancreatic cyst of enormous size simulating ascites 53
——— a case of acute bronchocele 236
——— a case of tachycardia (? Graves's disease) 237
PITT (G. Newton, M.D.), a case of combined sclerosis of the spinal cord 223
Plastic operation for rectal incontinence following modified Kraske's excision of rectum for cancer (J. J. Clarke) . . 195
Pneumonia, right lobar, complicating case of pneumococcal suppurating pericarditis (J. Fawcett, M.D., and F. J. Steward) . 46

PAGE

Poisoning from the external use of aniline oil (StClair Thomson, M.D.) 156
POWELL (Sir R. Douglas, Bart., K.C.V.O., M.D.), Presidential Address delivered at the Clinical Society of London on October 12th, 1900 lv
——— discussion on the epidemic of enteric fever among the troops in South Africa 100, 113, 129, 139, 143
Presidents of the Society since its formation xx
PRICE-JONES (C., M.B.) and PASTEUR (W., M.D.), a case of cancer of the stomach, with unusual vascular and blood changes 160
Pyramidal tract: injury to, in case of bullet wound of brain (W. G. Spencer) 238
Pyrexia, late syphilitic (intermittent essential fever of syphilis) (Campbell Williams) 28

Radiography, instantaneous (E. W. H. Shenton) 76
Raynaud's disease accompanying generalised scleroderma (H. D. Rolleston, M.D., and S. V. Pearson, M.D.) 215
Rectum: rectal incontinence, following modified Kraske's excision of rectum for cancer, treated by plastic operation (J. J. Clarke) 195
Reduplication, spurious, of second sound at apex of heart (T. Stacey Wilson, M.D.) 10
Regurgitation, aortic, probably due to syphilis (C. W. Chapman, M.D.) 64
——— aortic, apparently due to rupture of healthy valve (L. E. Shaw, M.D.) 220
——— ? patent ductus arteriosus regurgitation in case of congenital morbus cordis (P. Kidd, M.D.) 238
Resonance, normal thoracic, in left-handed persons (Sir H. Beevor, Bart., M.D.) 242
Retro-collic spasm (C. E. Beevor, M.D.) 245
Rheumatism, gonorrhœal, with bursæ filled with concretions of urate of soda (W. G. Spencer) 51
RICHARDS (J. Stewart, M.D.), a case of symmetrical tumours of the legs 227
——— a case of extraordinary curvature of the lower end of the femur following epiphysitis 231
Rigidity of muscles, localised (F. E. Batten, M.D.) 219
ROBINSON (H. Betham), a case of enlargement of the right foot ? cause 234

PAGE

Roentgen rays: instantaneous radiography (E. W. H. Shenton) 76
ROLLESTON (H. D., M.D.), a case of multiple cutaneous carcinomatosis after carcinoma mammæ 206
——— a case of progressive myositis ossificans (for F. W. Burton-Fanning, M.D.) 232
——— and PEARSON (S. Vere, M.D.), generalised scleroderma with Raynaud's disease 215
RUSSELL (J. S. Risien, M.D.), a case of myasthenia gravis . 216
——— and JOHNSON (Raymond), traumatic subdural hæmorrhage occasioning convulsions on the sixth day after the injury and successfully treated by operation 179

Sarcoma: central sarcoma of lower jaw: case shown three and a half years after enucleation (C. J. Symonds) 213
Scarlatina: nephritis without albuminuria; not preceded by scarlatina (W. P Herringham, M.D.) 34
Scleroderma, generalised, with Raynaud's disease (H. D. Rolleston, M.D., and S. V. Pearson, M.D.) 215
Sclerosis, combined, of spinal cord (G. Newton Pitt, M.D.) . . 223
SEGUNDO (C. S. de, M.D.), a case of chronic lymphangitis, by A. E. Bridger, M.D. (introduced by C. S. de Segundo, M.D.) . 229
SHAW (H. Batty, M.D.), a case of transfacial bullet wound . . 210
SHAW (Lauriston E., M.D.), a case of aortic regurgitation apparently due to rupture of a healthy valve 220
SHENTON (Edward W. H.), a paper on instantaneous radiography (introduced by J. H. Bryant, M.D.). 76
Sinus, frontal, empyema, chronic, radical cure (H. Tilley, M.D.) . 201
Skin: growths in skin in case of lymphadenoma (W. Lee Dickinson, M.D.) 246
Soda, urate of: bursæ filled with concretions of urate of soda in case of gonorrhœal rheumatism (W. G. Spencer) 51
SOLTAU (A. B., M.B.), a case of chronic hypertrophic osteoarthropathy (introduced by Percy Kidd, M.D.) 249
Spasm, retro-collic (C. E. Beevor, M.D.) 245
SPENCER (Walter G.), a case of gonorrhœal rheumatism, with bursæ filled with concretions of urate of soda 51
——— a case of bullet wound of the brain showing injuries to the optic nerve and the pyramidal tract, corresponding to the course and present position of the bullet 238
Spinal cord: degeneration, subacute combined, in association with pernicious anæmia (F. E. Batten, M.D.) 56

PAGE

Spinal cord, hæmorrhage into (case shown by W. A. Marris, M.D., for C. E. Beevor, M.D.) 212
Spinal cord: sclerosis, combined (G. Newton Pitt, M.D.) . . 223
Spleen: pronounced spleno-myelogenic leucocythæmia subsequent to an attack of malarial fever (D. Nabarro, M.D.) . 70
Splenectomy for wandering spleen (J. Bland-Sutton) . . . 31
STEWARD (Francis J.), a case of ureteral calculus simulating vesical calculus 149
——— and FAWCETT (John, M.D.), a case of pneumococcal suppurating pericarditis, treated by incision and drainage; right lobar pneumonia; death 46
STILL (G. F., M.D.), unusual case of obesity in a child (introduced by Dr. Dalton) 243
Stomach, cancer of, with unusual vascular and blood changes (W. Pasteur, M.D., and C. Price-Jones, M.B.) . . . 160
Strümpell's asthenic bulbar paralysis: see *Myasthenia* gravis pseudo-paralytica.
Subdural hæmorrhage, traumatic, occasioning convulsions; successful operation (R. Johnson and J. Risien Russell, M.D.) . 179
Sulphonal not causing hæmato-porphyrinuria (J. Calvert, M.D.) 41
Suppuration, acute, of thyroid gland complicating typhoid fever (R. J. Godlee) 189
SYMONDS (Charters J.), a case of central sarcoma of the lower jaw three and a half years after enucleation 213
——— dislocation of the patella 252
——— hæmorrhagic gummatous myositis 252
——— knee-joint eight years after erasion 253
Syphilis, aortic regurgitation probably due to (C. W. Chapman, M.D.) 64
——— late syphilitic pyrexia (intermittent essential fever of syphilis) (Campbell Williams) 28

Tachycardia: case of tachycardia (? Graves's disease) (S. Phillips, M.D.) 237
THIN (George, M.D.), a case of pronounced spleno-myelogenic leucocythæmia subsequent to an attack of malarial fever. By David Nabarro, M.D. (introduced by George Thin, M.D.) 70
THOMSON (StClair, M.D.), poisoning from the external use of aniline oil 156
——— a case of growth of the left tonsillar region . . . 203
——— a case of cystic hygroma of the neck, probably congenital 231